A PROSPECTIVE CLINICAL STUDY OF CENTRAL RETINAL VEIN OCCLUSION IN NON-DIABETIC INDIVIDUALS.

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CERTIFICATE

This is to certify that this dissertation entitled "**A PROSPECTIVE CLINICAL STUDY OF CENTRAL RETINAL VEIN OCCLUSION IN NON-DIABETIC INDIVIDUALS."** has been done under my guidance in the Department of OPHTHALMOLOGY, MADURAI MEDICAL COLLEGE, MADURAI.

I Certify regarding the authenticity of the work done to prepare this dissertation.

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DECLARATION

I, **Dr.D.HEMA**, Solemnly declare that the dissertation titled, "**A PROSPECTIVE CLINICAL STUDY OF CENTRAL RETINAL VEIN OCCLUSION IN NON-DIABETIC INDIVIDUALS"**has been prepared by me.

This is submitted to the "**THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY, CHENNAI**, In partial fulfillment of the requirement for the award of M.S., (Ophthalmology) Branch-III degree examination to be held in **APRIL 2013.**

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CONTENTS

PART – I

PART – II

RETINAL VASCULAR OCCLUSION

Venous occlusive diseases has been the most common retinal disease found in clinical practice. Depending on the involvement site and retinal perfusion extent, venous occlusions affecting the retina have been classified into 6 clinical entities differently,[1-4]

VENOUS OCCLUSIONS OF RETINA ARE CLASSIFIED AS:

1) Central Retinal Vein Occlusion (CRVO)

a) Non Ischemic or Venous Stasis Retinopathy

b) Ischaemic or Haemorrhagic Retinopathy

2) Branch Retinal Vein Occlusion (BRVO)

a) Major

b) Macular

3) Hemicentral Retinal Vein Occlusion (HCRVO)

a) Non Ischaemic

b) Ischaemic

About 50 % to 70 % of the patients with CRVO are above 50years of age and many have been reported to have an association with systemic Hypertension, Diabetes and cardiovascular disease.

ANATOMY OF RETINA

Retina, being a thin, and transparent membrane, is the inner part of the eyeball. And this tissue of the eye is the most highly developed structure. Retina, which has a surface area of 266 square mm, has an extension from the optic disc to oraserrata. Ophthalmoscopic examination when viewed grossly it is been divided into three regions[5,6] :

a) Optic disc,

b) macula lutea,

c) peripheral retina or general fundus.

CENTRAL RETINAL VEIN

The Central Retinal Vein seen on the optic nerve head is formed by the union of tributaries of retinal vein.The vein is enclosed in a fibrous envelope along with the artery in common and it courses on the lateral side with central retinal artery usually in its axial part.

The site where the vein exits from the optic nerve varies and it usually exits from the same aspect that of the nerve where the point of entry occurs with of the central retinal artery in about 42 % of the patients and the vein is found to be anterior to the artery in about 81 % .

The intra vaginal course is 3.8-8 mm and it is longer than of the artery with a greater fraction of the course in the dura mater.

TRIBUTARIES OF THE VEIN:

1. From the Retina.
2. From the posterior central vein.
3. From the optic nerve head including choroidal tributaries and from the peripapillary sclera at all levels.

Communications : -

The central retinal vein before it drains into inferior or superior ophthalmic veins and directly into cavernous sinus joins the orbital plexus of veins.

Blockage of blood flow does not occur in the central retinal veins when there is block in blood flow at cavernous sinus due to the multiple connections.

Structure of Central retinal vein

The central retinal vein which lies on the basement membrane has an endothelium which is continuous.Outside of this, it is a cell which may be a pericyte or a smooth muscle and the tunica media is represented as a smooth muscle cell that is separated from the underlying tissue. There is the presence of connective tissue adventitia in it.

CENTRAL RETINAL VENOUS OCCLUSION

Central Retinal Vein occlusion or among with its branches causes an ischaemic infarct of the retinal tissue that is already affected.

Two types of Central Retinal Vein Occlusion are clinically described[3,4,7] :

Central Retinal Vein Occlusion in the ischemic variety constitutes to 20 - 25 percent among all the cases while the major percentage of about 75 - 80% eyes with Central Retinal Vein Occlusion are of non ischemic variety.

Demographic characteristics:

Studies conducted variously have shown that males are more commonly affected than that of females[7,8]. The age of onset are variable, between 14 to 92 years[7-10]. The ischemic type more commonly occurs in people above 65 years while the nonischemic variety is of more common occurence in patients below than 45 years. The involvement risk of the fellow eye is of 5.6 - 6.6 percent in duration of 2.8 - 3.4 years in both the types of Central Retinal Vein Occlusion [4,7]. Developmental risk of both types of venous occlusion in the fellow eye is in range of 11.9 percent in a duration of 4 years[7].

Nonischemic variety can occur repeatedly in the same eye of 2.2 percent only with patients in duration of 5 years[7]

PATHOGENESIS:

Green along with Coinvestigators[11], described in their histopathological investigations, and accepted the hypothesis that formation of thrombus was the event primarily responsible for CRVO.

Proliferation of Endothelium and inflammation were the common associations, but they were believed to be in occurance in secondary to formation of thrombus rather than of primary events. The reason was unknown that whether the formation of thrombus occured in the region of Lamina cribrosa.

The formation of thrombus in the region of lamina cribrosa was the close anatomic association of the Central Retinal Vein with the Central Retinal Artery in this region as well as the narrow course of Central Retinal Vein through it contributes to the turbulence of flow and formation of thrombus [12].

A common adventitial sheath is shared by the central retinal artery along with the vein as they leave the head of optic nerve and passes along a narrow opening in the region of the lamina cribrosa.

Because of the narrow entrance in the region of lamina cribrosa, the vessels are in situated in a compartment which is tight and with a limited space for it to displace. This critical anatomical position predisposes to the formation of thrombus in the Vein by some major factors including slowness of blood stream, changes occurring in the wall of the vessel and blood changes.

Virchow's classical triad described in thrombus occurance are :

1. Slowing occurring in blood stream
2. Changes in the wall of the vessel.
3. Blood rheology Changes .

Klein along with Olwin[13] and Klein[14] , made postulations for the following mechanisms of pathogenesis that contributes to Central Retinal Vein blockage causing stagnation of flow in the vein and result in a primary thrombus occurance.

1. Blockage of the vein externally by the sclerosed central retinal artery, which is enclosed in same sheath and endothelial proliferation occurring secondarily.
2. Blockage by primary venous wall disease which is degenerative or inflammatory in origin.

3. A variety of factors produce haemodynamic disturbances like subendothelial atheromatous causes in the central retinal artery, spasm of the artery , arterial hypotension, dyscrasias of the blood, that is aggravated further by arteriosclerosis or anatomic relations which is unfavourable.

Hayreh [15], produced different types of retinal vascular blockage in rhesus monkeys, to establish the pathogenesis of Central Retinal Vein Occlusion.

Central Retinal Vein Occlusions lead to the stagnation of the blood in the retinal vein system and increased resistance in it causes stagnation of blood and also ischaemic damages to the retina.

It has been postulated that ischaemic damages to retina stimulated the VEGF production increase in the vitreous cavity.

The Increased levels in VEGF stimulated the neovascularisation of anterior and posterior segment that was responsible for the secondary complications in CRVO.

Also , VEGF can cause capillary leakage that will lead to macular edema [16] was shown , and this found to be the leading cause of visual loss in both ischaemic group and non ischaemic type in CRVO.

CLINICAL CHARACTERISTICS SEEN IN ISCHEMIC AND NON ISCHEMIC GROUP OF CRVO:

	Clinical Characteristics	ISCHEMIC CRVO	NON-ISCHEMIC CRVO
1.	Vision	Usually it is markedly impaired.	mildly impaired.
2.	Relative Afferent Pupillary Defect	Present	Absent
3.	Visual Fields examination	Peripheral fields-Abnormal	Normal - peripheral fields.
4.	Fundus Fluorescein Angiography	Extensive Capillary Non-perfusion -More than 10 disc areas	Nil or minimal Capillary Non-perfusion.
5.	Neovascularisation seen	Present	Not present
6.	Electro Retinogram	Decreased b wave amplitude and decreased b/a ratio.	Normal b wave amplitude and Normal b/a ratio.

RISK FACTORS AND ASSOCIATIONS OF RETINAL VEIN OCCLUSIONS ATTRIBUTED[17,18,19] :

SYSTEMIC DISEASES AND CENTRAL VEIN OCCLUSION:

Certain systemic diseases can influene the formation of the thrombus in the central retinal vein :

A) External compression .

B) Primary formation of thrombus through disorders of the blood.

C) Inflammatory and degenerative conditions of the vein itself.

EXTERNAL COMPRESSION:

Hypertensive or arteriosclerotic changes in the central retinal artery could compress the vein as they pass through the region of lamina cribrosa through a common sheath.(13)When this process is exacerbated by stagnation of the venous blood, a secondary thrombus forms and this leads to the occlusion of the vein. Other causes of external compression includes papilledema, papillitis , graves disease , orbital space occupying lesions and retro- bulbar intra nerve sheath injections.(18, 64)

HAEMORRHEOLOGICAL FACTORS :

Various factors in the constituents of blood can predispose to formation of thrombus in the blood such as high viscous nature of the blood, increased coagulability and reduced thrombolytic state. Increased viscous nature of the blood associated with raised haematocrit values has been reported in patients with retinal vein blockage and had been compared with controls(27).

PRIMARY DISEASE IN THE VESSEL WALL :

Infectious causes of vasculitis like syphilis and AIDS and systemic vascular diseases like connective tissue disorders have an association with occlusion of the retinal vein(37) .Localised inflammations of the vein could lead to occlusion of the vein like in sarcoidosis and the diseased wall could interact with the clotting factors leading to the formation of the thrombus.

1). Systemic vascular diseases : –

Diabetes Mellitus, systemic Hypertension, Insufficiency Of carotid artery.

2). Ocular Diseases : –

Glaucoma of Open Angle type, Ischaemic Optic Neuropathies, Drusen of the Optic Nerve head and Pseudo tumour cerebri.

3). Haematologic Alterations:–

Syndromes due to Hyperviscosity, Dysproteinemias, Multiple Myeloma, Blood dyscrasias like Polycythemia Vera, Lymphoma, Sickle Cell diseases, Anaemia, Elevated plasma homocysteine, deficiency of factor xii, Antiphospholipid antibody syndrome, Activated protein C resistance, deficiency of Protein C, deficiency of Protein S.

4). Inflammatory and Auto immune Vasculitis : –

Autoimmune mediated disorders like Systemic lupus erythematous.

5). Medications : –

Oral Contraceptives, Diuretics and Hepatitis B Vaccine.

6). Infective Vasculitis :–

HIV and Syphilis, Herpes Zoster, Sarcoidosis.

7). Others: –

After retrobulbar block, Dehydration, during Pregnancy.

ASSOCIATED MEDICAL CONDITIONS s:

1). Hypertension : -

Hypertension is found to be one of the most common cause of CRVO occurring in older patients.

The eye disease Casecontrol study [20] found that an increased risk occurred in any type of Venous occlusive disease in those patients with Systemic Hypertension and DM.

Persons with non-perfused Venous occlusive disorders had an odds-ratio that is greater, implicating where in systolic and diastolic Hypertension are risk factors compared to a perfused Venous occlusive disease. Similar associations were found with the other prospective and retrospective cross-sectional studies with Systemic Hypertension.

2). Diabetes Mellitus :-

One of the other leading cause of CRVO found in adults was Diabetes which caused microvascular angiopathy and thrombo-embolic phenomenon. Several authors[21,22,23] have

reported the incidence of diabetes in patients with CRVO to found to vary from 13 – 34%.

Prevalence of Diabetes mellitus is found to be more in individuals with a non-perfused central retinal vein blockage than matched controls from large population database study.

3). Hyperlipidemia :-

Association of hyperlipidemia with central retinal vein occlusion has been reported. The incidence has been found to be low in previous reports[24,25] .

More emphasis has been laid recently, on detecting hyperlipidemia in these patients as the incidence is constantly increasing. Raised cholesterol Patients are put under high risk group.

4). Hypercoagulability : -

Haematological abnormalities, conditions particularly that predispose to a hypercoagulable state, has been identified in persons with central retinal vein blockage. Individuals below 60 years of age were found to have a greater association with increased coagulable states and inflammatory nature,

compared with that of older age group with a higher incidence of systemic vascular disease risk factors.

Lahey with his investigators found only one lab value outside normal limits in fifty-five patients that contributed to 27 %, younger than 56 years, suggesting the systolic high coagulable states[26].

Studies have demonstrated that an increased incidence of coagulation cascade abnormalities was found including resistance of protein C ,factor P. Leiden presence , presence of antiphopholipid antibodies and fibrinogen levels that was abnormal. [27,28,29,30, 31].

In a meta-analysis of studies that was published, an association was found with raised plasma homocysteine and low serum folate levels, but not with serum Vit. B12 levels and the thermo labile methyl tetrahydrofolate reductase genotype[32] and same was observed with increased viscosity from blood dyscrasias ,dysproteinemias and dehydration in central retinal vein occlusion[33] .

5). Migraine : -

Friedman MW [34] in his study found that nine percent of patients who had headache was due to migraine and speculated that it was related to platelet abnormality.

Fong et al[35] in his series, observed that 4 out of 102 (4%) cases and that of Walton, spalton, found 2 out of 17 (12%) of the patients suffered from migraine.

6). Mitral Valve Prolapse:-

Gorden et al[10], reported in his study ,7 out of 11 patients with occlusion of central retinal vein showed evidence of Mitral Valve Prolapse. The authors suggest that hyperactivity of the platelets is the leading cause of central retinal vein occlusion in these patients.

7). Collagen Vascular Disorders:-

Collagen Vascular Disorders are seen in association with retinal vascular disorders[25,36], of which SLE is known to

cause an inflammatory reaction in blood vessels including those of retina.

8). AIDS : -

Teiaer and Sonnaberd[37] reported on a case of central retinal vein occlusion in an AIDS patient and postulated that it might be due to high levels of anticardiolipid antibody , immune complex deposits or a toxic direct effect of HIV virus on the endothelium and thus perivasculitis may be a part of non – infective AIDS retinopathy. Others have also reported occlusion of central retinal vein in association with AIDS[18].

9). Carotid Artery Disease : -

Brown et al[38] demonstrated atherosclerosis of the Carotid Artery by Digital Substraction Angiography in 3 patients with Ischemic type of central retinal vein occlusion. Lazzarro[10], reported 6 out of 12 patients can have central retinal vein occlusion .

10). Medications : -

a) Oral Contraceptives : -

Many reports have shown a trend towards hypercoagulability with the usage of oral contraceptives, Kulvin et al[10] performed an experimental study on monkeys with oral contraceptives and observed the dilatation of Central retinal vein. Stowe [39] et al showed the occurrence of intimal proliferation with occlusion in patients who took oral contraceptives for about 4 years.

b) Diuretics :-

Gutman[18] suggested that patients who received diuretics may be at risk of developing central retinal vein blockade due to secondary heamoconcentration effect that lead to hyperviscosity.

OCULAR CONDITIONS:-

Magargal et al[40] reported 16 out of 69 (25%) of their patients had associated ocular abnormalities.

a). Glaucoma :-

Glaucoma usually of primary open angle type, is well known to predispose to central retinal venous occlusion in patients of elder age group. Incidence of chronic open angle glaucoma seen in cases of central retinal vein occlusion has been reported to range from 66 to 75 %. Mansour et al [59] and Strabhlman et al[10] found that no relationship existed between cup-disc Ratio and central vein occlusion.

b). Other ocular conditions that caused deformation or mechanical pressure on the optic nerve head and lamina cribrosa were Ischemic Optic Neuropathy, congenital abnormality of Nerve head, drusen of the Optic Nerve head, Optic disc traction syndrome and pseudotumor cerebri, and these were found to have associations with Central Vein Occlusion.

c) External compression of the globe and also optic nerve from thyroid related Ophthalmopathy or mass lesion or a patient with head trauma and orbital fracture may also result in Central Vein Occlusion.

NATURAL COURSE SEEN IN CENTRAL RETINAL VEIN OCCLUSION

The retinopathy of Central Retinal Vein blockage generally is self-limiting and gets resolved over a period from weeks to years. Some of these eyes are likely to develop the following complications:

1). Macular Edema:

Macular dysfunction is seen to occur in most of the eyes with Central Retinal Vein Occlusion, that leads to central visual loss. In eyes with nonischemic type of Central Retinal Vein Occlusion, the resolution of macular microcystic edema can be slow, without any residual loss of visual acuity [(4).]

In few patients,the macular edema in chronic duration can progress to multiple permanent structural changes in macula like cystoid macular degenerations , macular pigmentary disturbances and epiretinal membrane.

2). Ocular Neovascularization:

This is known to be one of the serious complication seen in ischemic type of Occlusion of Central Retinal Vein. The site of common occurence is the iris and the angle then followed by the optic disc and peripheral retina. About onethird of the eyes with iris neovascularisation was found not to develop neovascular glaucoma.

The reported incidence overall of Neovascular Glaucoma in ischemic variety of Central Retinal Vein Occlusion was found to vary from 14 - 60 percent widely.[4, 41-44]. However, it is seen that every eyes with ischemic variety of CRVO do not develop ocular neovascularisation but the risk is very high in the first seven months after the onset of the disease . It is observed in some patients that who had severe pain due to neovascular glaucoma at the initial visit had a history of visual loss three to four months before. This condition was named as “100 days glaucoma” and these patients typically presented with corneal edema,elevated intra-ocular pressure,severe neovascularisation of the iris, and closed angles on gonioscopic examination.

The study of Hayreh et al suggested that severity and nature of extent of retinal ischemia were the principal factors, though the follow-up duration also played a role in the incidence of ocular Neovascularization. However, it is also known that 23 to 42 percent of eyes with Central Retinal Vein block might have a pre-existing glaucoma or hypertension[45-47]. Neovascular glaucoma was not reported in non ischemic type of Central Retinal Vein Occlusion, unless when it was associated with an underlying diabetic retinopathy, carotid artery disease, or any other retinopathy with extensive capillary blockage.

3). Vitreous Haemorrhage:

Vitreous hemorrhage in Central Retinal Vein blockage was found to have an incidence of up to 7 percent[42,44]. Neovascularization was found not as the only cause of vitreous hemorrhage, and the other common causes were the leakage of blood through internal limiting membrane[4] of retina in the early stages of Occlusion. The haemorrhage can also occur sometimes due to intra – retinal abnormalities in the microvasculature secondary to occlusion of the vein and due to posterior vitreous detachment.

4). Cilioretinal Artery Occlusion:

One of the major contributing factor of severe visual loss in nonischemic type of Central Retinal Vein Occlusion was the associated cilioretinal artery occlusion. This might have caused a permanent field defect, which can be sectoral or centrocecal, depending on nature of the extent of involvement[(4)].

REVIEW OF LITERATURE

1). Prevalence and Risk factors of Occlusion of Retinal Vein in an Asian population.

Br. J. Ophthalmol 2008; 92:1316-1319.

L.L.Lim, N. Cheung, J.J Wang et al[48], conducted a population based, cross-sectional study, the Singapore Malay Eye study consisting of 3280 (78.7%) malay adults aged 40 to 80 years .The results of the study showed that Retinal Vein Occlusion was associated with raised systolic levels of blood pressure in individuals with a history of angina, or increased total cholesterol levels and heart attack.

2).

Paul Mahoney, Wong DT, Ray JG et al[49], systematically reviewed all studies between January 1985 and July 2007 that compared cases with any form of Retinal Vein Occlusion with controls. They generated pooled odds ratio (OR) and estimates of the population with attributable risk percentage for systemic hypertension, hyperlipidemia and diabetes mellitus.

The results observed from the twenty-one studies, including 2916 patients and 2864 controls, showed that both hypertension (OR, 3.5; 95%

Confidence interval [CI] 2.5 – 5.1) and hyperlipidemia [CI, 1.7 – 3.7] were significantly associated with any form of Retinal Vein Occlusion.

The percentage of cases with any form of Occlusion of Retinal Vein that was attributed to hypertension were 47.9% (95% CI, 31.2% - 63.1%)TO Diabetes Mellitus was 4.9% (0,8% - 11.5%) and to hyperlipidemia 20.1 % (, 5.9% - 43.8%).

Thus, the study concluded that hypertension and hyperlipidemia are the most common risk factors in adults for Retinal Vein Occlusion and diabetes mellitus is lesser . It also concluded that lowering of blood pressure and / or Serum lipid levels can improve the visual acuity or the complications of Retinal Vein Occlusion has to be determined.

3). Ocular Neovascularisation Associated with both CRVO and HCRVO.

Retina 32:1553-1565, 2012

Hayreh SS, Zimmermann et al[(50)], in their study on 912 (673 Non-ischemic type and 239 Ischemic type)cases with CRVO and HCRVO investigated the incidence of ocular neovascularisation in CRVO and hemi central vein occlusion. Ophthalmic evaluation was done at initial

and every visits which included the testing of vision, examination of fields, thorough anterior segment, fundus examinations and fundus fluorescein angiography.

The results of the study also showed that in ischemic variety of crvo, within 6 months duration from time of onset, the probability of development of iris neovascularisation was 49 percent, neovascularisation at the angle is 37 percent, neovascular glaucoma 29 percent, and disc neovascularisation 6 %. More severe peripheral retinal haemorrhages were significantly associated with Iris Neovascularisation (P is 0.005); Angle Neovascularisation (P is 0.0004) and Neovascular Glaucoma (P is 0.012).

This study finally concluded that in Ischemic type of Central Retinal Vein Occlusion, neovascularisation of the anterior segment was much more common than posterior segment Neovascularisation and the cumulative chances of developing the change is maximum within the first 6 months.

4). Natural History of the visual outcome in CRVO.

Ophthalmology vol :118 . no.1 January 2011.

Hayreh SS et al, Patricia A ,Podhajsky et al[51], investigated the natural history of visual acuity outcome in Central Vein Occlusion, and systematically reviewed in six-hundred sixty seven consecutive patients with 30 patients having both eyes resulting in 677 eyes with Central Retinal Vein blockage from 1973 till 2000.

Visual evaluvation was carried out in all patients by recording vision using Snellen's acuity chart, assessing the visual fields with a Goldmann perimeter. The same ophthalmic evaluation was performed at each consecutive visit. Occlusion of Central Retinal Vein was classified as Non-Ischemic variety with 588 eyes and Ischemic variety with 109 eyes at an initial visit based on the functional and morphologic data.

The results of the study showed that within the first three months, visual acuity was 20/100 or better 78 percent in Non-ischemic cases of Central Retinal Vein block and only on 1 percent with Ischemic cases of Central Retinal Vein Occlusion (P was 0.0001) and visual defects were minimal in 91 percent and 8% (P is 0.0001).

Final visual acuity was 20/100 or better in 83percent of cases on resolution of Macular edema in Non-ischemic variety of Central Retinal Vein Occlusion, and only 12% with Ischemic type of Central Retinal Vein Occlusion (P was 0.0001) and defects in visual field was minimal or mild in 95 percent and 18% (P is 0.0001). on resolution of macular edema, eyes with initial visual acuity of 20/70 or worse, vision improved in 59 percent with Nonischemic type of Central Retinal Vein block, with no significant improvement in Ischemicvariety of Central Retinal Vein Occlusion (P is 0.55).

Also, with resolution of Macular edema, eyes with moderate to severe defect in visual field, improvement was seen in 86 percent of Non-ischemic type of Central Retinal Vein block, but no improvement was seen in eyes with Ischemic variety of Central Retinal Vein Occlusion (P is 0.83).

In Nonischemic variety of Occlusion of Central Retinal Vein , development of final pigmentary degenerative changes, epiretinal membrane formation and or both was the main cause of poor final visualoutcome. This showed that initial presentation and the final visual acuity outcome in 2 types of Central Retinal Vein Occlusion are entirely different.

The study concluded that differentiating central Vein Occlusion as Non-ischemic and Ischemic types, are based on the functional criteria and was mandatory and fundamental in determining the visual outcome. Visual acuity outcome was good in Nonischemic type and poor in Ischemic type of Central Retinal Vein Occlusion.

5) .Natural history of the Central Retinal Vein Occlusion-: An evidence based systematic review approach.

Ophthalmology 117, No.6 June 2010.

McIntosh, Sophie L, Rogers, Ning Cheung et al[52], systematically reviewed case series for 5 years to describe the natural history of Central Vein Occlusion.

Two investigators identified all the observational studies that evaluated the natural history of Retinal Vein Occlusion and all clinical trials evaluating interventions for Central Vein Occlusion, an untreated control arm was included.

This study reviewed 5966 citations, also 53 studies providing 3271 eyes with Central Vein Occlusion for the analysis of its natural history.

Visual outcome was poor at baseline (20/40) and decreased further over time.

Although some studies reported an improvement in Visual acuity, none of these improvements resulted in visual outcome, better than 20/40. Upto 34 percent of the eyes with Nonischemic type converted to Ischemic variety of Retinal Vein Occlusion over a 3 year period. In Ischemic Retinal Vein Occlusion cases, Neovascular glaucoma developed in 23 percent of eyes in 15 months. In Non-ischemic variety, , macular edema was resolved in 30% of eyes approximately over time, and subsequent neovascular glaucoma was rare in occurance. Thus the study concluded that untreated eyes with Central Vein Occlusion had in general poor vision and it declined further over time. One quarter of eyes with Nonischemic variety was found to convert to Ischemic-variety of Central Retinal Vein Occlusion.

6) .Retinal Vein Occlusion.

Indian Journal of Ophthalmology 1994;42:109-132.

In this study, Hayreh SS[(53)], has discussed about the recent advances and classification of Retinal Vein Occlusion, the pathogenesis and demographic characteristics of various types of Retinal Vein Occlusion; differentiating Nonischemic type and Ischemic type of

CRVO based on six entities like testing vision, examination of visual fields, Relative afferent pupillary defect, Electroretinogram, Opthalmoscopy and Fundus Fluorescein Angiography; the course and management of the various types of Retinal Vein Occlusion.

7) . Atherosclerotic and thrombophilic risk factors found in patients with Ischemic CRVO.

Retina 2011 April;31(4):724-729.

Sodi, Giambene B, Marcucci R, Sofi F et al[54], studied in one hundred and three patients with acute CRVO that was unilateral in presentation with 41 Ischemic type and 62 Non-ischemic type.. The frequency of traditional cardio-vascular risk factors were analysed and plasma levels of a variety of thrombophilic markers were measured.

The results of the study showed that arterial systemic Hypertension, Hypercholesterolemia, Postmethionine hyperhomocysteinemia and decreased Folic Acid levels were more frequent in occurance in patients with Ischemic entity CRVO than in Non-ischemic entity.(P is 0.030; P is 0.011; $P<0.001$; and $P=0.044$ respectively)

The risk factors for Ischemic type were arterial Hypertension (odds ratio [or], 3.32;; P=0.037) ,Hypercholesterolemia (OR, 3.03; 95% CI, 1.06 – 8.65; P is 0.042), decreased folic acid levels (OR, 6.77; 95% CI, 1.59-28.79; P is 0.011).

The study concluded that some atherosclerotic and thrombophilic risk factors may increase the risk of an Ischemic type of CRVO.

8) . High Lipoprotein (a) levels association with an increased risk of Retinal Vein Occlusion.

Atherosclerosis 2010 May; 210(1):278-281

Sofi F, Marcucci R, Fedi S et al[(55)], compared 262 patients with an median age of 66 years (5-88); 122 males and 140 females , with 261 age and sex comparable healthy individuals.

They found that circulating concentrations of Lipoprotein (a) were found to be of significantly difference in patients when compared to healthy subjects - 189 (60-1898) mg per L vs 119.5 (6-1216) mg per L ; P less than 0.0001 respectively . No significant difference was observed with in relation to different types of occlusion whether Central or Branch Occlusion.. In the Univariate analysis,Lipoprotein (a) levels more than

300 mg per L were found to be associated with an higher risk of Retinal Vein Occlusion (OR: 2.39, 95 percent CI, 1.39 – 3.59; P less than 0.0001). Then three models of multi-variate analysis were performed. In all the models, Lipoprotein (a) levels more than 300 mg per L confirmed its role as a risk factor for Retinal Vein Occlusion . first model, OR: 2.15 (95 percent CI; 1.39 to 3.32), P is 0.001 ; second model, OR: 3.11 (95 percent CI 1.77 to 5.62), P less than 0.0001; third model, OR: 3.48 (95 percent CI, 1.88 to 6.43), P less than 0.0001]

Thus, this study reported that in a large population with Vein Occlusion patients, high concentrations of Lipoprotein (a) were significantly related to Occlusion which was independent from the other traditional and emerging risk factors, suggesting that they also might play a role in the pathogenesis.

9) . Risk factors in central retinal vein occlusion.

Oftalmologia. 2011;55(2):27-37.

Calugaru D et al[56], in his study observed that risk factors for the occurrence of central vein occlusion were to a limited extent similar to those of cardiovascular diseases like arteriosclerosis, systemic

hypertension, diabetes mellitus and dyslipidemia. Hyperhomocysteinemia was found to be a part of an essential risk factor for arteriosclerosis intervening directly with the local mechanism that caused venous and arterial occlusions. Ocular hypertension and glaucoma were risk factors and significantly associated with pathogenesis of central retinal occlusion. Therapy with anticoagulants and platelet antiaggregating drugs exposed the patient for development of central vein occlusion influencing the visual outcome adversely without having any evidence of the protective or beneficial effects.

10).Etiology of the central retinal vein occlusion.

Oftalmologia. 2011;55(2):12-26.

Calugaru D et al[57], In his another study has shown that primarily arteriosclerosis was responsible for the majority of pictures of central vein occlusion occurrence. The venous occlusions that occurred during the development of the known disorders already represented venous occlusions secondarily. In young adults central retinal vein occlusion represented a nonspecific change in general that contributed to the emergence of Multifactorial causes from a number

of individual. Even In unusual cases of central vein occlusion the etiology was known but within the vast majority of patients the specific cause, even the causes that contributed to the occurrence of this disease remains unknown, due to the limitations of ophthalmic literature and the scarcity of histopathologic material.

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AIMS AND OBJECTIVES

1. To study the risk factors and systemic associations of CRVO in non-diabetic individuals.
2. To compare the demographic factors (like age, sex) in nonischemic variety and ischemic variety of CRVO.
3. To compare the final ocular outcome in ischemic and non-ischemic CRVO.
4. To study the natural course of CRVO.

MATERIALS AND METHODS

A prospective clinical study was carried out to analyse the clinical course of CRVO in non-diabetic individuals.

All suspected venous occlusion were screened at retina clinic of Govt. Rajaji Hospital for a period of one year.

72 patients with vein occlusion were seen, out of which 40 eyes of 40 patients satisfying the inclusion criteria were enrolled in the study.

The patients were in the age group of 35 years to 75 years. There were 30 males (75%) and 10 females (25%) in the study group.

The right eye was involved in 18 patients (45%) and left eye in 22 patients (55%).

Thorough medical history and general examination of the patient was carried out to rule out the presence of any systemic disease predisposing to a vein occlusion. In doubtful cases, expert opinion was obtained. A detailed ocular history which included history of any previous ocular disease and the treatment was elicited.

TABLE : 1.

Inclusion criteria :

i) Confirmed presence of CRVO.

ii) Non-diabetic individuals with CRVO.

iii) Patients of all ages presenting with CRVO.

TABLE : 2

Exclusion criteria:

i. Presence of Branch Retinal Vein Occlusion (BRVO), Diabetic retinopathy or any other retinal vascular disease or with vitreous haemorrhage.

ii. Previous photocoagulation done for retinal vascular disease.

iii. Any other external eye disease likely to affect the visual acuity during period of study.

Baseline investigation was carried out immediately and thorough lab workup plan was carried as follows:-

TABLE 3 :

LAB - WORK UP PLAN.

- Haemoglobin.
- Complete blood count (CBC).
- Fasting Blood Sugar levels.
- Post-prandial Blood Sugar levels.
- Lipid Profile.
- Blood Urea, Serum Creatinine.
- Erythrocyte Sedimentation Rate (ESR).
- Mantoux test.
- Blood VDRL for Syphilis.
- Coagulation Studies.
- ELISA for HIV.

Electrophysiological test:

- Electrocardiogram
- Echo cardiogram.
- Carotid Doppler for selected patients.

Ocular examination at the time of initial presentation of the patient included the following :

i) Best corrected visual acuity.
ii) Pupillary reaction
iii) Slitlamp biomicroscopy.
iv) Indirect Ophthalmoscopy.
v) Fundus Fluorescein Angiography. (F.F.A.)

The appearance of disc edema unilaterally with dilatation and tortuousity of major blood vessels of retina and varying amounts of retinal haemorrhage were said to have Central Retinal Venous Occlusion.

After the initial visit, patient was followed up every month for a minimum period of 6 months.

The criteria chosen to classify the patient in ischemic and non-ischemic includes the following:-

i) Poor visual acuity.
ii) Presence of Relative Afferent Pupillary Defect. (RAPD).
iii) Fundus showing extensive haemorrhages and cotton-wool spots.
iv) Fundus Fluorescein Angiography showing non-perfusion areas greater than 10 disc diameters.

Those patients with Ischemic Central Vein Occlusion who developed neovascularisation of iris out of neovascularisation of disc and elsewhere were treated with pan retinal photocoagulation in two sittings while the remaining patients were followed up at monthly intervals upto the conclusion of the study.

RESULTS

40 eyes of 40 patients who satisfied the eligibility criteria were included in the prospective study on the etiology and course of CRVO.

- The study group consisted of 30 males (75%) and 10 females (25%); the male: female ratio being 3: 1.

GENDER STUDY GROUP

TABLE 1

	Frequency	**Percent**
Male	30	75
Female	10	25
Total	40	100

TABLE 2 :

Number of cases	Non-ischemic CRVO		Ischemic- CRVO	
	FREQUENCY	PERCENT	FREQUENCY	PERCENT
MALES	20	71.4%	10	83.3%
FEMALES	8	28.6%	2	17.7%
TOTAL	28	100.0%	12	100.0 %

- Most of the patients in our series were found to be males contributing to 83.3% in ischemic variety and 71.4% in the nonischemic variety with females contributing to 28.6 % of the nonischemic patients and 17.7% of ischemic group in the total.
- In our study Non- ischemic CRVO patients are about 70% and Ischemic-CRVO patients are about 30% of the total.

AGE – DISTRIBUTION

AGE - STUDY GROUP :

TABLE 3 :

AGE (YEARS)	FREQUENCY	PERCENT
35 - 44	2	5.0
45 - 54	10	25.0
55 - 64	18	45.0
65 - 74	10	25.0
TOTAL	40	100.0

Demographic study shows that majority of patients are in the age group of 55 – 75 years (70%) with mean age = 58.3 years ; SD = 8.28; in the study group.

INITIAL PRESENTATION

TABLE 4 :

AGE	NON-ISCHEMIC - CRVO		ISCHEMIC- CRVO	
	FREQUENCY	PERCENT	FREQUENCY	PERCENT
35 - 44	2	7.15 %	0	0
45 - 54	9	32.14 %	1	8.3 %
55 - 64	14	50 .0 %	4	33.3%
65 - 75	3	10.71 %	7	58.3%
TOTAL	28	100.0	12	100.0

- Majority of the patients with Non-ischaemic CRVO were around 55 to 64 years of age at initial presentation contributing to 50%
- Most of the patients with Ischaemic CRVO were between 65 to 75 years of age, contributing to 58.3 %

III. LATERALITY :

TABLE 5 :

	FREQUENCY	PERCENT
RIGHT EYE	18	45 .0
LEFT EYE	22	55 .0
TOTAL	40	100 .0

In our observation , all our study cases were unilateral with the right eye affected in 18 cases (45 %) and left eye in 22 cases (55 %).

COMPARISION OF THE LATERALITY OF THE EYE AFFECTED IN NON ISCHEMIC GROUP, ISCHEMIC AND CONVERTED GROUP

TABLE 6:

	NON-ISCHEMIC CRVO		ISCHEMIC- CRVO		CONVERTED	
	NUMBER	PERCENT	NUMBER	PERCENT	NUMBER	PERCENT

RIGHT EYE	14	58.4%	3	25%	1	25%
LEFT EYE	10	41.6%	9	75%	3	75%
TOTAL	24	100.0	12	100.0	4	100.0

In our study , right eye was affected in 14 patients of non- ischemic group contributing to 58.4 % and 3 patients in ischemic variety with 25 %. The left eye was affected in 10 patients of non- ischemic group and 9 patients of ischemic group. Thus in comparing the laterality, right eye was more affected in non- ischemic group and left eye in ischemic group.

IV . DISTRIBUTION OF TYPE OF VENOUS OCCLUSION :

TABLE 7:

	FREQUENCY	PERCENT
NON-ISCHEMIC CRVO	24	60.0
ISCHEMIC CRVO	12	30 .0

CONVERTED	4	10.0

In our study series, among the 40 patients , 28 of them presented with non-ischemic variety contributing to 60 % of the total and ischemic variety contributing to 30% in the total distribution.

Out of the 28 patients in the non-ischemic group, 4 of them converted to ischemic variety at the end of the study.

V. CLINICAL MANIFESTATIONS :

TABLE 8:

S.NO	CLINICAL MANIFESTATIONS	NUMBER	PERCENT
1.	SUDDEN PAINLESS LOSS OF VISION	25	62.5%

2.	BLURRING OF VISION	17	42.5%
3.	RAPD	8	20.0%
4.	AMAUROSIS FUGAX	3	7.5%
5.	FLASHES OF LIGHT,/FLOATERS	13	32.5%

The major presenting symptom among our patients were sudden painless visual loss contributing to 62.5 % forming the major feature and blurring of vision to 42.5 %. Other manifestations include amaurosis fugax and flashes of light , floaters.

VI. VISUAL ACUITY :

TABLE 9 :

S.No	VISUAL ACUITY	NON-ISCHEMIC CRVO	
		Initial	Final
1.	6/6 – 6/9	4	4

2.	6/12 – 6/18	10	12
3.	6/24 – 6/36	12	8
4.	<6/60	2	0
5.	CFCF or WORSE	0	0
Total Number		28	24

In non-ischemic group, visual acuity at the initial presentation was good with 4 patients having 6/6 – 6/9 ; 10 patients with 6 /12 – 6/18 and 12 patients with 6/24 – 6 /36. At the end of the study , majority of them showed no change in visual acuity.

VISUAL ACUITY IN ISCHEMIC GROUP

TABLE : 9 A

S.No	VISUAL ACUITY	ISCHEMIC CRVO

		Initial	Final
1.	6/6 – 6/9	0	0
2.	6/12 – 6/18	0	0
3.	6/24 – 6/36	0	2
4.	<6/60	8	6
5.	CFCF or WORSE	4	4
Total Number		12	12

In the ischemic group , majority of the patients at the initial presentation had a poor visual acuity of less than 6/60. At the end of the study , 2 patients showed improvement to 6/24 - 6/ 36 , and 6 of them showed no change in visual acuity.

VISUAL ACUITY IN CONVERTED GROUP

TABLE : 9 B

S.No	VISUAL ACUITY	CONVERTED	
		Initial	Final
1.	6/6 – 6/9	0	0
2.	6/12 – 6/18	0	0
3.	6/24 – 6/36	2	0
4.	<6/60	2	3
5.	CFCF or WORSE	0	1
Total Number		4	4

In the converted group , 2 patients at the initial presentation who had visual acuity of 6/24 – 6/36 , at the end had visual acuity of less than 6/60. The final visual acuity in this group was poor.

TABLE 10 :

S.		NON-ISCHEMIC CRVO	ISCHEMIC CRVO

NO	CRITERIA	FREQUENCY	PERCENT	FREQUENCY	PERCENT
1.	SHOWED IMPROVEMENT IN VISUAL ACUITY (2 LINES OR MORE)	4	16.7 %	2	12.5 %
2.	NO CHANGE IN VISUAL ACUITY	16	66.6 %	6	37.5 %
3.	WORSENED (2 OR MORE LINES)	4	16.7 %	8	50.0 %

ASSOCIATED MEDICAL CONDITIONS

TABLE 11:

SL. NO	MEDICAL CONDITIONS	NUMBER	PERCENTAGE
1.	Systemic Hypertension	16	40.0%
2.	Hyperlipidemia	6	15.0%
3.	Chronic Renal Failure	4	10.0%
4.	Coronary Artery Disease	1	2.5%
5.	Migraine	1	2.5%
6.	Anemia	2	5.0%
7.	Cerebro vascular Accident	2	5.0%

A history of associated medical condition was elicited in all our patients. Out of which 16 patients had Systemic Hypertension which contributed as the single largest cause among them.

Of the 16 patients, 3 patients were not on regular anti-Hypertensive therapy; 4 patients were on therapy but not on adequate dosage; 2 patients were detected having Hypertension after the onset of CRVO.

COMPARISION OF FREQUENCY OF HYPERTENSION IN NON ISCHEMIC AND ISCHEMIC - CRVO

TABLE 12:

S.NO	CASES WITH HYPERTENSION	NON-ISCHEMIC CRVO	ISCHEMIC -CRVO	TOTAL	P-VALUE
1.	YES	9 (32.1%)	7 (58.3%)	16	0.1213
2.	NO	19 (67.9%)	5 (41.7%)	24	
		28 (100.0%)	12 (100.0%)	40	

P VALUE = 0.1213 .This is not stastically significant.

There is no statistically significant proportion in hypertension between Ischemic and Non Ischemic patients (P value = 0.1213).

ASSOCIATED OCULAR CONDITIONS

TABLE 13:

SL. NO	OCULAR CONDITIONS	PERCENTAGE
1.	Primary Open Angle Glaucoma	5.0%
2.	Tilted Optic Nerve Head	2.5%
3.	Optic Nerve Head Drusen	0%

In our study, 2 patients who presented with Non-Ischemic CRVO had Primary Open Angle Glaucoma .

RESULTS OF LAB STUDIES

TABLE 14:

SL. NO.	LAB TESTS	PERCENTAGE
1.	High Serum Cholesterol	15.68%
2.	Elevated Renal parameters	7.5%
3.	High ESR	9.8%
4.	Low Haemoglobin	5.85%

Results of Lab investigations showed that patients above the age of 40 years had a high level of Serum Cholesterol (more than 200 mgs%) than the people below 40 years.

OCULAR SEQUELAE

TABLE 15:

SL. NO.	OCULAR SEQUELAE	PERCENTAGE
1.	Macular Oedema	48.0%
2.	Macular Degeneration	28.92%
3.	Hard Exudates	7.69%
4.	Sheathing	4.85%
5.	Collaterals	19.0%
6.	Neovascularisation of Iris	12.5%
7.	Neovascularisation of Disc	5.0%
8.	Neovascularisation elsewhere	7.6%
9.	Neovascular Glaucoma	4.69%

- The major ocular sequelae seen in non-ischemic CRVO patients was macular edema .
- Neovascularisation of Iris was seen in 5 patients with Ischemic CRVO and constituted 12.5% of all patients in the study.
- Patients belonging to Non-ischaemic CRVO did not develop neovascular complications during the study.

CAUSES OF POOR VISUAL RECOVERY

TABLE 16:

SL. NO	CAUSES	PERCENTAGE
1.	Macular Oedema	48.09%
2.	Macular Degeneration	28.60%
3.	Macular Hard Exudates	21.42%
4.	Vitreous Haemorrhage	1.92%

- Macular Oedema was the major contributing factor for poor visual recovery among our patients.

DISCUSSION

This study was a non-randomised prospective study including 40 patients with CRVO conducted in a tertiary care hospital at Madurai.

The aim of the study was to determine the risk factors and associated medical conditions of CRVO in non-diabetic individuals. All the patients in the study met the inclusion criteria.

AGE DISTRIBUTION:

The majority of the patients in our study were in the age group of 45 – 65 years accounting for 65% of the total.

In a study conducted by Hayreh et al[51] , from 1973 through 2000, in 667 patients ,he found that majority of patients of Non-Ischaemic CRVO (559 patients) were in the age group of 65 years and older, contributing to 47% and patients with Ischaemic CRVO in the age group of more than 65 years contributed to 75%.

Andrew K.Vine et al [(60)], in his study on 74 cases found that mean age of presentation of CRVO was 69 years. Paul O'Mahoney[49], retreived all studies published between January 1985 and July 2007 and found that most cases presented between ages 60 and 65 years.

In our study, the patients with Non-Ischemic CRVO at initial presentation were in the age group of 55 to 64 years contributing to 50% and patients more than 65 years contributed to 28.6% which was lower than the study by Hayreh et al. Patients with Ischemic CRVO in the age group of more than 65 years contributed to 58.3% in our study, which is concordant with his study.

SEX DISTRIBUTION:

Males and females were studied for their distribution in relation to CRVO.

Based on analysis of 32 studies collected and reviewed by McIntosh et al[52], they found an increased incidence of CRVO in males contributing to 56% (1549 eyes) and 44% females (1230 eyes). It was in concordance with our study, where male predominance is seen than the females.

Male to female ratio in our study was 30: 10, giving a ratio of 3.0:1.0 which is slightly lower than that reported by Fong et al[35] and Frucht. J. Yanko[25] .

Thus a male predominance was seen in CRVO than in females and this may be due to the increased predisposing factors seen in males.

LATERALITY:

CRVO is typically unilateral. In the present series, right eye was affected in 18 patients and left eye in 22 patients which is quite different from earlier reported series where the right eye was frequently involved as described by Hart.

But study conducted by Hayreh et al[51], Shows that right eye was more affected in non-ischemic and left eye in ischemic CRVO individuals which was the same in our study as right eye in non-ischemic was 58.4% than 41.6 % in left eye, in ischemic CRVO, left eye was 75 % and right eye in 25 %.

Demographic and clinical characteristics: (Hayreh et al[51] – Natural History of Visual outcome in CRVO)

SL. NO	EYE INVOLVEMENT	NON ISCHEMIC VARIETY (n = 559 eyes)	PATIENTS WITH ISCHEMIC VARIETY (n = 109 eyes)	CONVERTED GROUP (n = 48 eyes)
1.	Right	266 (48%)	45 (42%)	18 (39%)
2.	Left	265 (47%)	62 (57%)	27 (59%)
3.	Both	29 (5%)	1 (1%)	1 (2%)

Recent studies have shown an equal incidence. In our series, the left eye involvement was slightly higher.

CLASSIFICATION OF CRVO

Central Venous Occlusion is classified into 2 major types by Hayreh et al[53], based on the combined data acquired from six tests: vision, visual fields examination, relative afferent pupillary defect, electro-retinography, ophthalmoscopy, and fundus fluorescein angiography.

1) NonIschemic CRVO or Venous Stasis Retinopathy and

2) Ischemic CRVO or Haemorrhagic Retinopathy.

Leber et al[61], in his study, also classified CRVO into two entities as Non- Ischemic CRVO and Ischemic CRVO.

CONVERSION OF NON-ISCHEMIC TO ISCHEMIC CRVO:

McIntosh et al[52], based on the analysis of 6 studies, (675 eyes) reported adequate quality data on the conversion of Non-ischemic to Ischemic CRVO. The incidence of such conversion rate from Non-ischemic to Ischemic CRVO ranged from 0% to 27% over a period of 10 weeks to 13 months post CRVO.

In study by Hayreh et al[51], which included 588 eyes with Non-ischemic CRVO 48 eyes converted to Ischemic type; There was a higher prevalence of history of Cerebro Vascular Diseases in those that converted as ischemic from nonIschemicvariety. (P = 0.006). Visual changes after conversion to Ischemic CRVO were similar to those seen in eyes with Ischemic CRVO as first diagnosis.

In our study, out of 28 patients of Non-ischemic CRVO, 4 patients converted to Ischemic CRVO, contributing to 14.28%.,this is in concordant with the studies observed by Mc intosh et al and Hayreh et al.

VISUAL ACUITY:

McIntosh et al[52], based on the review of 53 studies in 3271 eyes with CRVO found that – For all CRVO cases, including Non ischemic CRVO, baseline visual acuity was generally poor (<20/40) and majority of studies reported a mean decrease in visual acuity over time. Ischemic CRVO had poor vision at presentation and follow up. Ischemic CRVO had a mean baseline visual acuity of 9 letters and an average decrease of 35 letters over time compared with Non-ischemic CRVO cases, which had a mean baseline visual acuity of 31 letters and decrease by only 3 letters beyond 12 months.

Quinlan et al[36], in a retrospective study of 107 Non-ischemic and 61 Ischemic CRVO eyes (using a criterion of more than and equal to 5 disc diameter of capillary non-perfusion) and a follow up of 6 months to 6 years, mean 22 months reported 15% had an improvement of 3 lines or more from the baseline and 31% lost 3 lines or more.

In all eyes with Ischemic CRVO, initial visual acuity was 20/100 or less, the visual acuity at the end of the study was 20/200 or less in 93%, counting fingers or less in 54%, hand movements or less in 36%.

Chen et al[62], in a case series of 59 eyes with Non Ischemic CRVO followed up for at least 1 year (average 2.5 years) found that visual acuity improved by 2 lines or more in 15%, remained stable in 56 % , and decrease by 29 %. They concluded that Non ischemic CRVO frequently resulted in significant permanent visual loss. They also found that initial visual acuity had no predictive value in prognosis.

Zegarra et al [41] in his study which he followed up for 1 - 8 years in 25 eyes, found that in 10 eyes with Non-ischemic CRVO, the visual acuity at the end was 20/30 or better in 50% and 20/60 in 30%; whereas among Ischemic CRVO eyes, 82% had final visual acuity of 20/400 or worse.

In our study, in Non-Ischemic CRVO, 4 patients had initial visual acuity of 6/6 to 6/9, 10 patients had 6/12 to 6/18, 12 patients had 6/24 to 6/36 and 2 patients had less than 6/60. At the end of the study 4 patients showed an improvement in 2 lines or more, contributing to 16.7% while 66.6 % showed no change in visual acuity and 4 patients had visual acuity that worsened by 2 lines or more. Our study results is in concordant with the study by Chen et al[62] and Zegarra et al[(41)].

In Ischemic CRVO, 8 patients had an initial visual acuity of <6/60 and 4 patients had counting fingers close to face. At the end of the study, 2 of our patients (12.5%) showed an improvement , 6 patients (37.5%) showed no changes in visual acuity, and 8 patients (50.0%) had visual acuity worsening of 2 lines or more. This results are in concordance with the studies done by Zegarra and Quinlan et al.

In the converted group, all patients had a final poor visual acuity of less than 6/60.

The primary cause of poor visual outcome in Non-ischemic CRVO is due to Macular Oedema, while in Ischemic CRVO, Retinal Ischemia was the major factor and Macular Oedema was minor cause.

In a recent SCORE (Standard Care Vs Corticosteroid for Retinal Vein Occlusion) study[(63)] in which CRVO eyes were combined into one

group without any differentiation into Ischemic and Non-ischemic CRVO, in the 73 eyes with out any treatment, visual acuity improved in 26%, remained same in 19% and deteriorated in 55% at 12 months follow up.

NATURAL COURSE OF CRVO:

In a study by Hayreh et al[51], the median time for Macular edema to resolve, was 23 months in those with Non-ischemic CRVO and 29 months in those with Ischemic CRVO as first diagnosis. Overall, for the Non-ischemic CRVO group with initial visual acuity 20/70 or worse, improvement was seen in 59% in which edema resolved; compared with 26% in which edema was still present. In contrast, among eyes with Ischemic CRVO as first diagnosis, and initial visual acuity of 20/70 or worse, there was no significant association of the presence or the absence of Macular edema with improvement in visual acuity (P=0.55)

McIntosh et al[52], based on the analysis of 7 studies (159 eyes) reported the development and resolution of Macular edema over time.

In cases with Ischemic CRVO, the proportions that described resolution of Macular edema ranged from 0% to 73% over time ranging

from 2 to 15 months post CRVO. In cases with Non-ischemic CRVO, the corresponding proportion was approximately 30%. In Non-ischemic variety, Macular edema was found to resolve in nearly 30% of patients over time.

Hayreh et al[50], in a prospective study of 78 eyes with ischemic CRVO ,found development of any type of neovascularisation in 66.7% (iris NV in 57.7%,NVG in 33.3%,angle NV in 47.4 %,disk NV in 1%) This study showed that the development of anterior segment new vessels was maximum in the first 6 months and minimal after that . Browning et al[64], in his prospective study on 105 eyes with ischemic crvo , found that 32 % developed anterior segment neovascularisation , 12 % angle NV without iris NV .This was also reported in 1983 by a prospective study of 78 eyes with ischemic CRVO.

In another study by Hayreh et al[50], in a study comprising of 912 subjects (673 non-ischemic and 239 ischemic) CRVO, he found the probability of development of Neovascularisation of iris was 49 percent, new vessels at the angle is 37 percent, NV Glaucoma 29 percent, retinal NV 9%. The conclusion of the study was anterior segment neovascularisation was more common than posterior segment neovascularisation.

In our study, Macular edema was the major ocular sequelae, contributing to 48%. contributing to poor visual recovery in patients with Non-ischemic CRVO. Neovascularisation of iris was seen in 12.5% and Neovascularisation of the disc in 5.%.This is in concordant with the study conducted by Hayreh et al ,and Browning et al[64], where anterior segment neovascularisation was more common than the posterior segment but the proportion was low , this may be due to small sample size observed in our study.

Management :

The therapeutic regimens advocated and tried in Hayreh et al study includes Anticoagulants, Fibrinolytic agents, Low Molecular Weight Dextran, CO_2 Inhalation, Vasodilators, Hyperbaric Oxygen, and Systemic Corticosteroids were used for resolution of Macular edema. Another conclusion derived from the study was Non-ischemic CRVO resolved spontaneously over time and no specific treatment was advocated as done in our study.

ASSOCIATED MEDICAL CONDITIONS :

Mahoney et al[49], systematically retrieved all studies published between January 1985 and July 2007, and estimated attributable risk percentage for Systemic arterial Hypertension, Diabetes Mellitus and Hyperlipidemia. Of the 21 studies including 2916 cases and 28646 controls, both Hypertesion (Odds Ratio 3.5) and Hyperlipidemia (Odds ratio 2.5) were significantly associated with Retinal Vein Occlusion occurring in any forms.. The percentage of cases with any form of Retinal Vein Occlusion attributed to Hypertension was 47.99% (95% CI) to Diabetes Mellitus 4.9% (95% CI) and to Hyperlipidemia was 20.1% (95 % CI).

Lim et al[48], in their Singapore Malay eye study in 3280 adults (40 – 80 years) found that Retinal Vein Occlusion was associated with higher systolic blood pressures (age – adjusted odds ratio), higher total cholesterol and LDL values.

Sodi et al[54], in their study on 103 patients who had CRVO that was unilateral in presentation was (41 Ischemic and 62 Non-ischemic cases) and assessed the percentage of traditional Cerebro Vascular risk factors and found that Arterial Hypertension, Hypercholesterolemia, Elevated factor VIII and reduced Folic Acid levels were more frequent in patients with Ischemic CRVO than in Non-ischemic CRVO.

Hayreh et al[51], in his study showed that the Ischemic CRVO group also had a higher prevalence of arterial Hypertension (p=0.052) and Diabetes Mellitus (p=0.0180) compared with Non-ischemic CRVO.

In our study, Hypertension contributed as one of the major systemic risk factors for CRVO contributing to 40 % (16 cases) which is in concordant with studies observed by Mahoney et al[49], and Lim et al[48] , but the proportion of systemic hypertension in non- ischemic CRVO (9 cases)was slighty higher than ischemic CRVO(7 cases), than observed in other studies. In this study, P VALUE OF (ARTERIAL HYPERTENSION) was found to be = 0.1213, which is not stastically significant and this might be due to the smaller sample size as well as due to lesser duration of study.

SUMMARY

Central Retinal Vein Occlusion is one of the Venous occlusive disease affecting the retina. In this study, 40 eyes of 40 patients who satisfied the inclusion criteria were observed.

- Non-ischemic CRVO was observed in 28 patients and Ischemic CRVO in 12 patients: 4 patients from Non-ischemic CRVO converted to Ischemic CRVO. Majority of CRVO cases were Non-ischemic CRVO which was observed in most of the studies and also in our study.
- Majority of the patients in Non-ischemic CRVO were in the age group of 55 – 64 years in our study where as the study by Hayreh et al[51] have shown majority of Non-ischemic cases above 65 years. In ischemic- CRVO ,most of the patients were above 65 years which was the same in the studies by Hayreh et al.
- Male predominance was seen in a ratio of 3.0: 1; but it was slightly lower than reported by Fong et al.
- CRVO is typically unilateral in presentation. All our cases were unilateral, no bilateral CRVO was reported in our study.

- In our study, the initial and final visual acuity was generally good in Non-ischemic cases where as the initial visual acuity in Ischemic group was below 6/60. The Ischemic and Converted patients had visual acuity that was poor at the end of the study, even though the initial visual acuity was good in the Converted group, the final overall visual acuity was poor. Many studies conducted by Hayreh et al[51], McIntosh et al[52], observed the same results.
- In our patients, the systemic risk factors associated with CRVO were Systemic Hypertension, Chronic Renal Failure, Hyperlipidemia and Cerebro Vascular Accidents. Out of which Hypertension contributed as the major risk factor. Singapore Malay eye study[48], also observed a higher association of Systolic Blood Pressure with CRVO. Mahoney et al[49], found that both Hypertension and Hyperlipidemia were significantly associated with any form of Retinal Vein Occlusion.
- Major ocular sequelae noted in our study was Macular edema, Macular degeneration, Neovascularisation of Iris and Optic disc.
- Macular edema noted in 48% of our cases, contributed as a major cause of poor visual outcome in these cases. The same ocular sequelae was observed in studies conducted by Hayreh et al[50] and McIntosh et al[52]. Neovascularisation of iris contributed to 12.5 %

and neovascularisation of disc and else where as 5.0 % and 7.5 %, Suggesting anterior segment neovascularisation more common than posterior segment. Our results were in concordant with the studies done by Hayreh et al and Browning et al[64].

CONCLUSION

- This study demonstrates that,Non-ischemic CRVO contributes to the majority of cases of CRVO.
- Males are more commonly affected than females.
- Non-ischemic CRVO was seen more commonly in the age group of 55-64 years while Ischemic CRVO more than 65 years.
- The initial visual acuity in Non-ishemic CRVO was generally good; and majority of cases show no change in visual acuity through out the study.

  Macular edema accounted for the subset of visual morbidity in these patients.

  Macular edema resolved spontaneously over a period of time in Non-ischemic cases.
- In Ischemic CRVO, the initial and final visual acuity were poor. The major ocular sequelae in these cases were neovascularisation of iris and optic disc.
- Systemic medical conditions seen in our patients were Hypertension, Hyperlipidemia and Chronic Renal Failure with

systemic Hypertension contributing to increased frequency among the illness. So thorough systemic evaluation has to be done in patients with CRVO, more than 40 years.

- Adequate initial control of high blood pressure and cholesterol levels are essential.
- Regular systemic, ophthalmologic follow up are important in early diagnosis of the disease as well as monitoring the progression of the disease.
- It is important for all patients with systemic illness like hypertension , hyperlipidemia ,coronary artery disease and chronic renal failure to undergo ophthalmological evaluvation regularly since early diagnosis and regular follow-up can reduce the deterioration of vision occurring due to the ocular sequelae in these patients.

GENDER STUDY GROUP

CHART 1

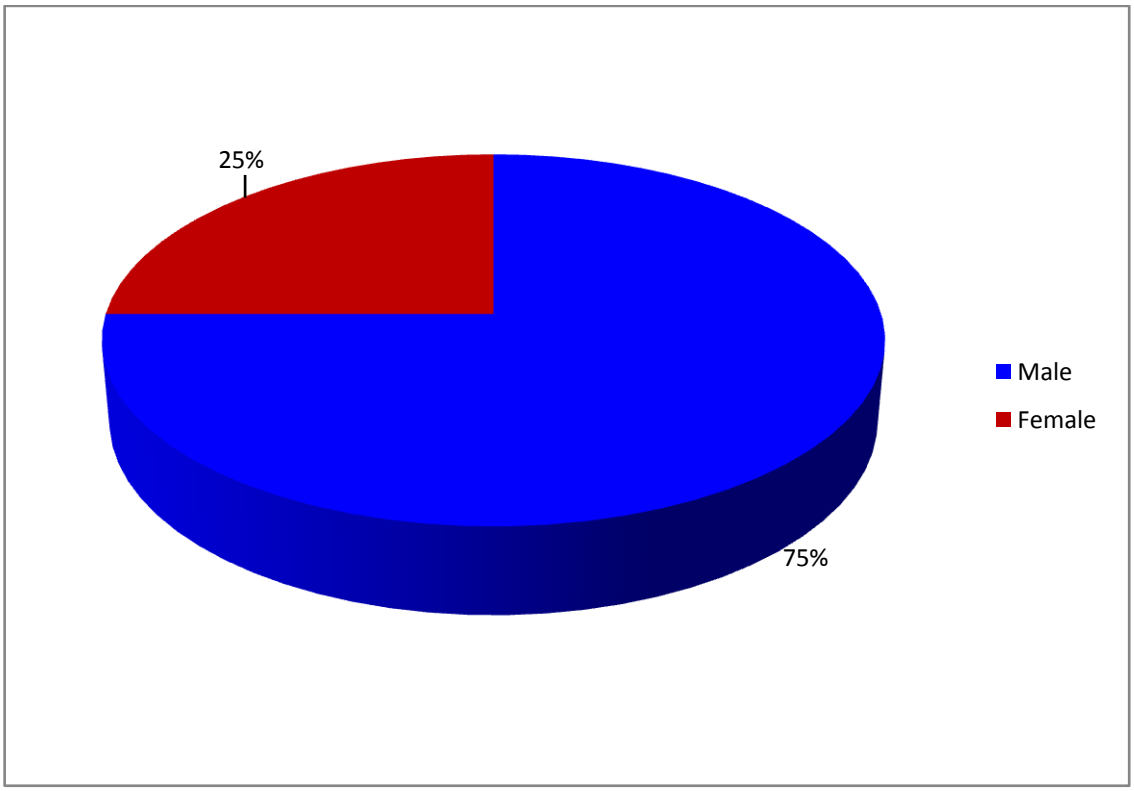
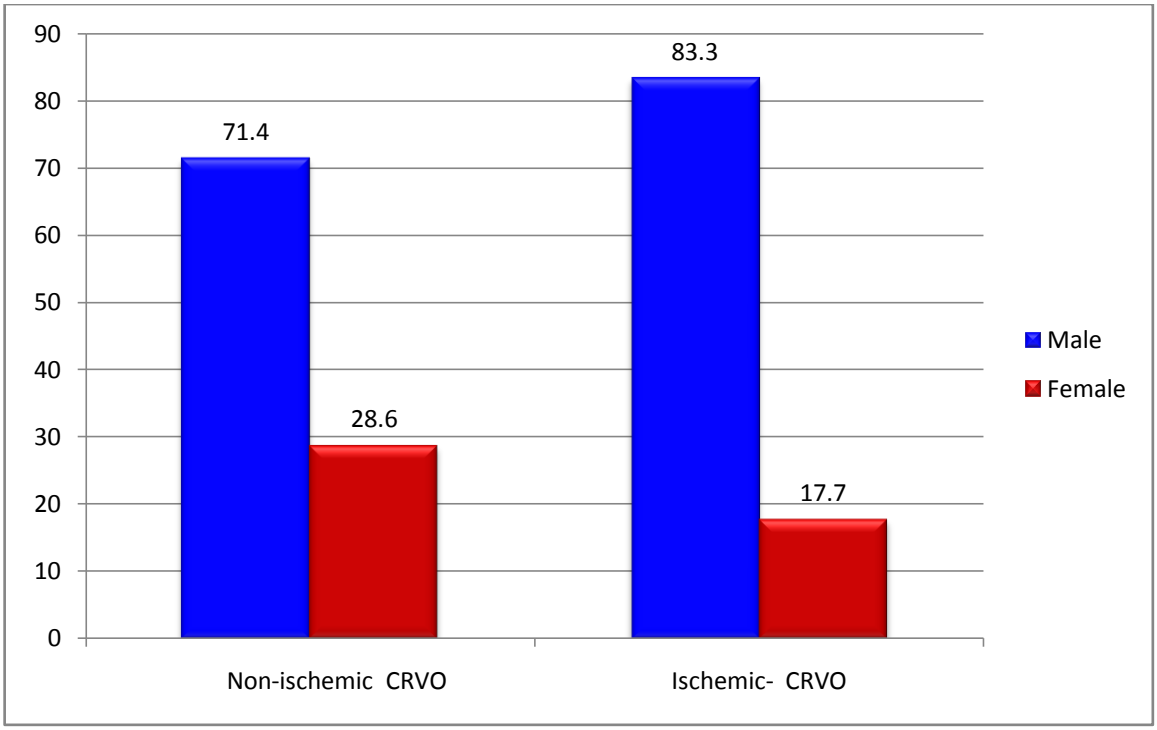
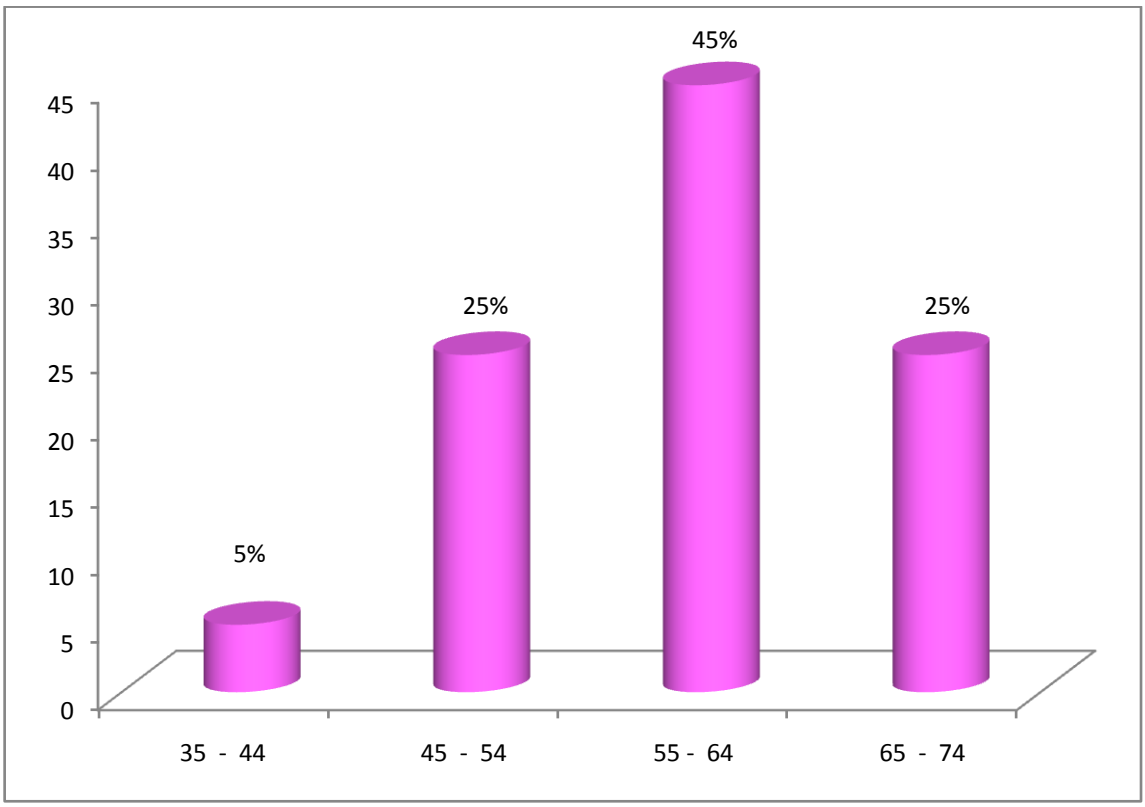


CHART 2


90
80
70
60
50
40
30
20
10
0
71.4
28.6
83.3
17.7
Non-ischemic CRVO
Ischemic- CRVO
Male
Female

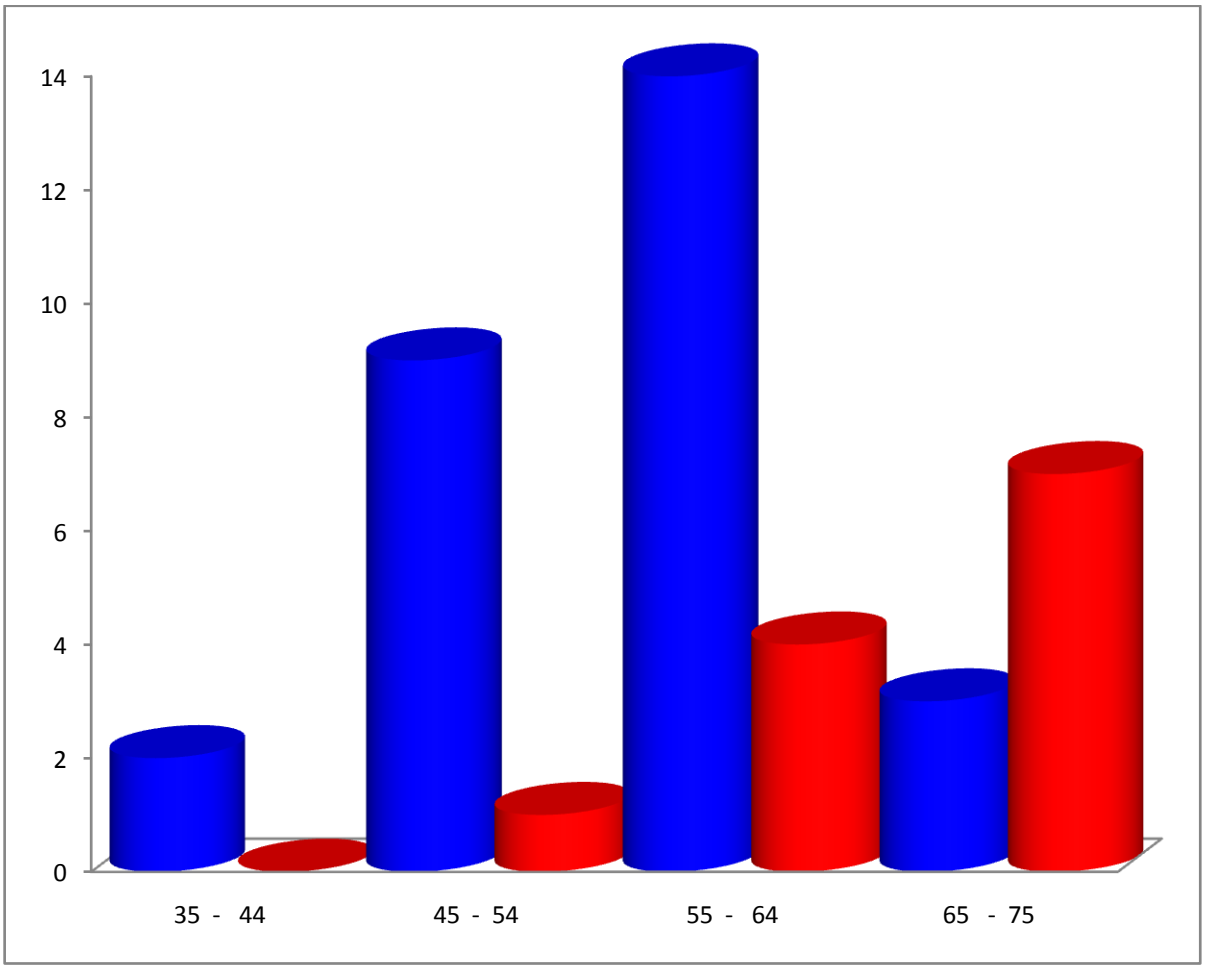
AGE - STUDY GROUP

CHART 3



INITIAL PRESENTATION

CHART 4



LATERALITY

CHART 5

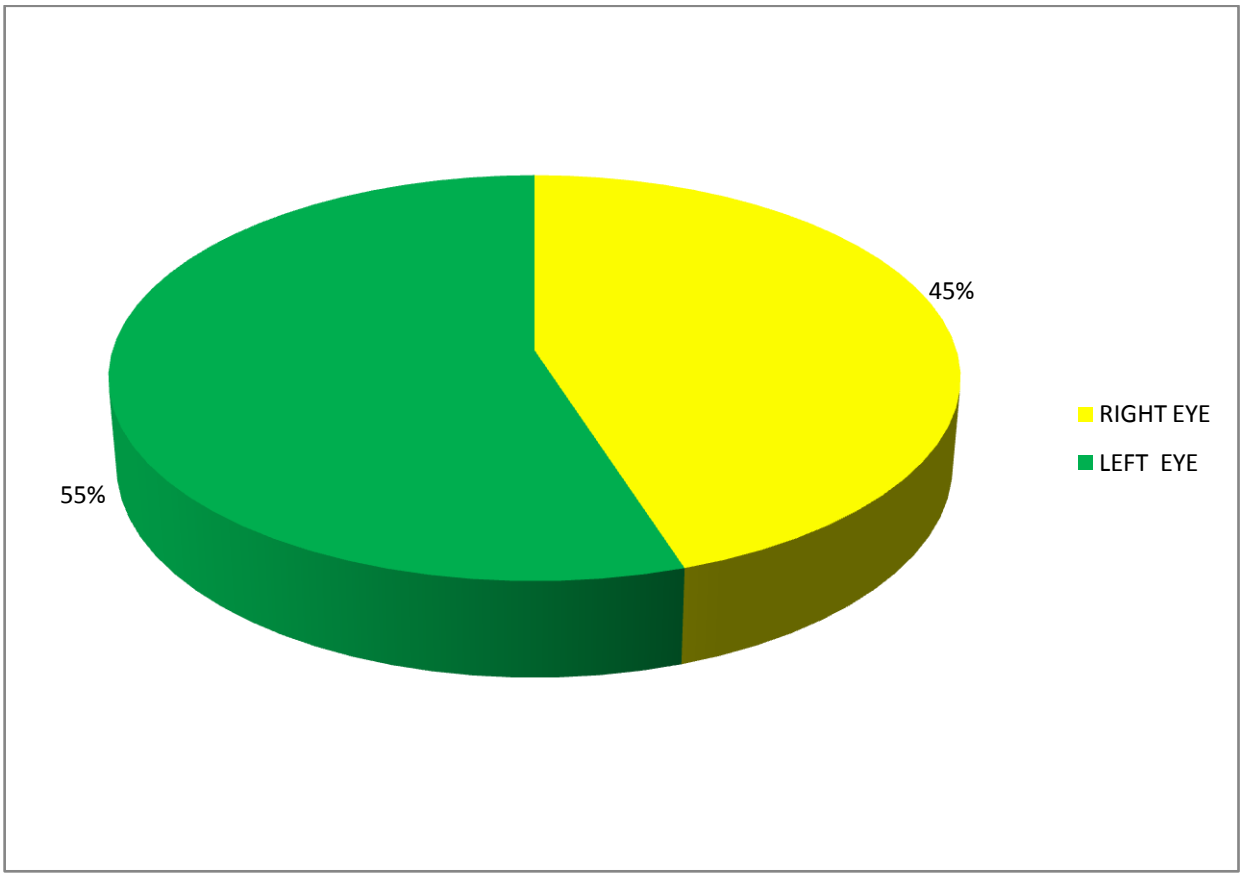
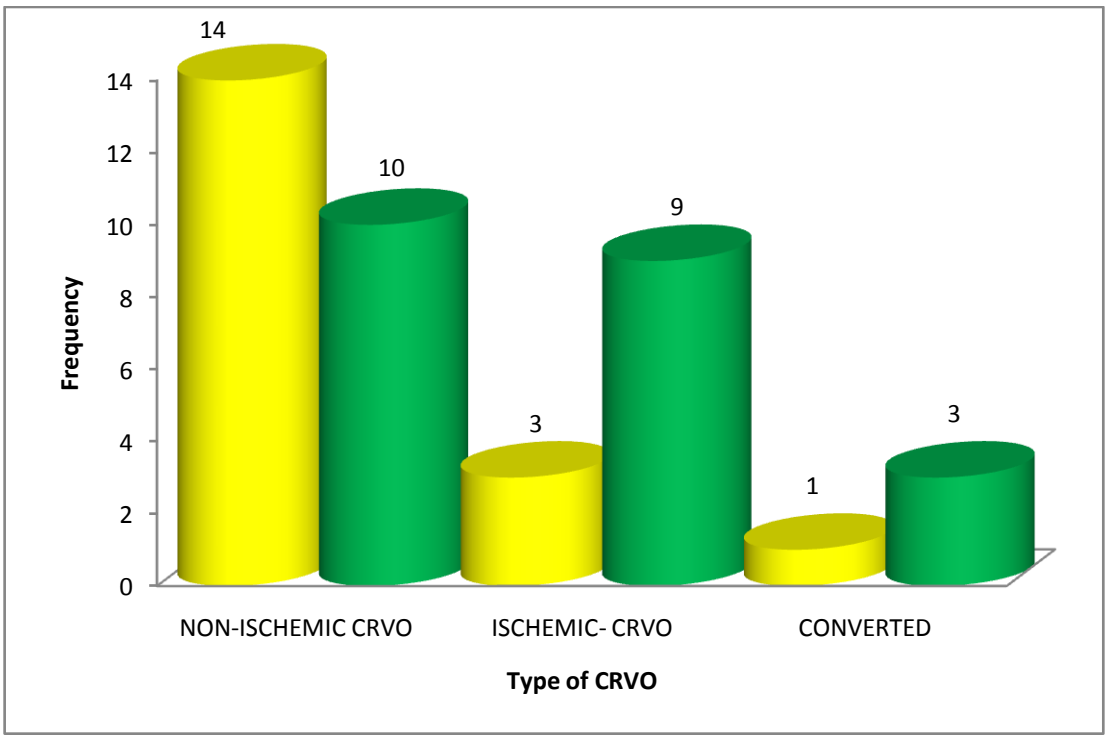
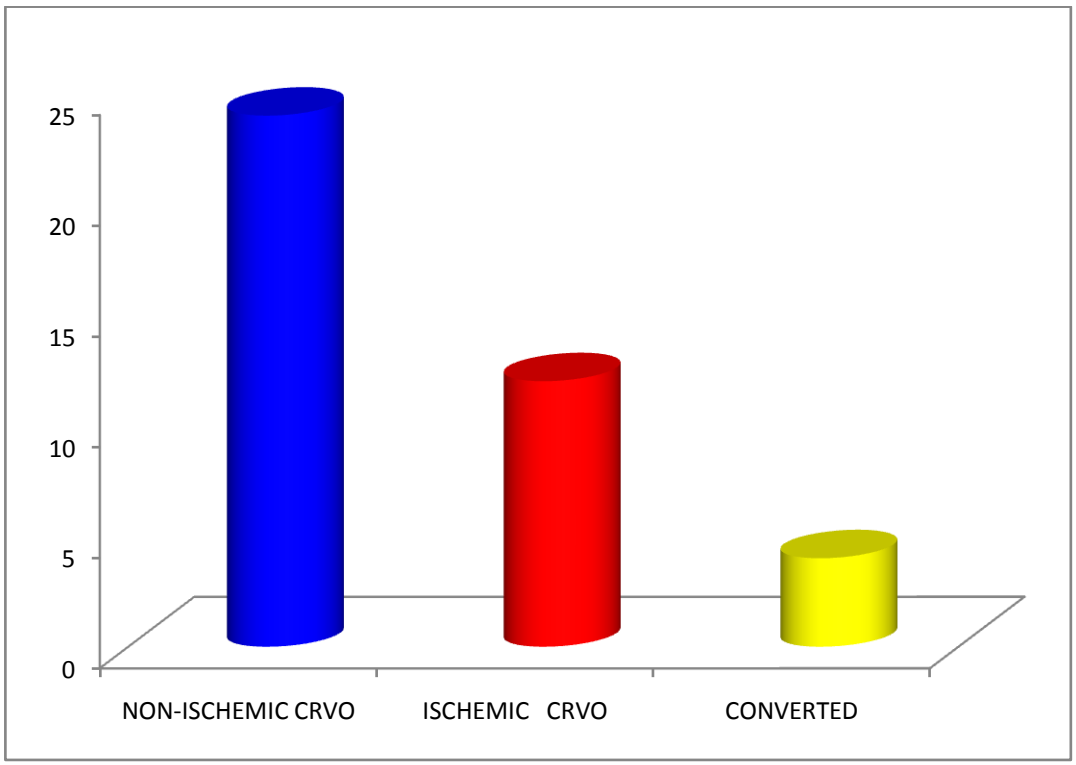


CHART 6


14
10
3
9
1
3
14
12
10
8
6
4
2
0
Frequency
NON-ISCHEMIC CRVO
ISCHEMIC- CRVO
CONVERTED
Type of CRVO

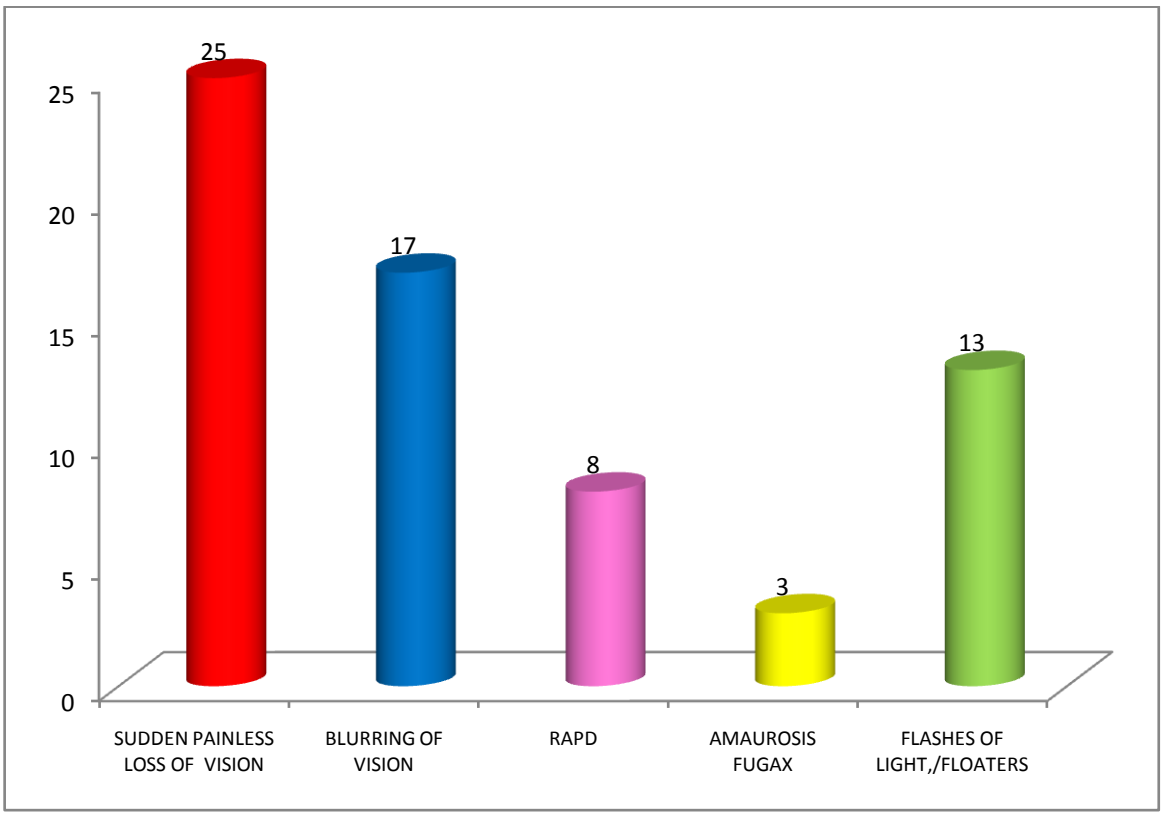
DISTRIBUTION OF TYPE OF VENOUS OCCLUSION

CHART 7



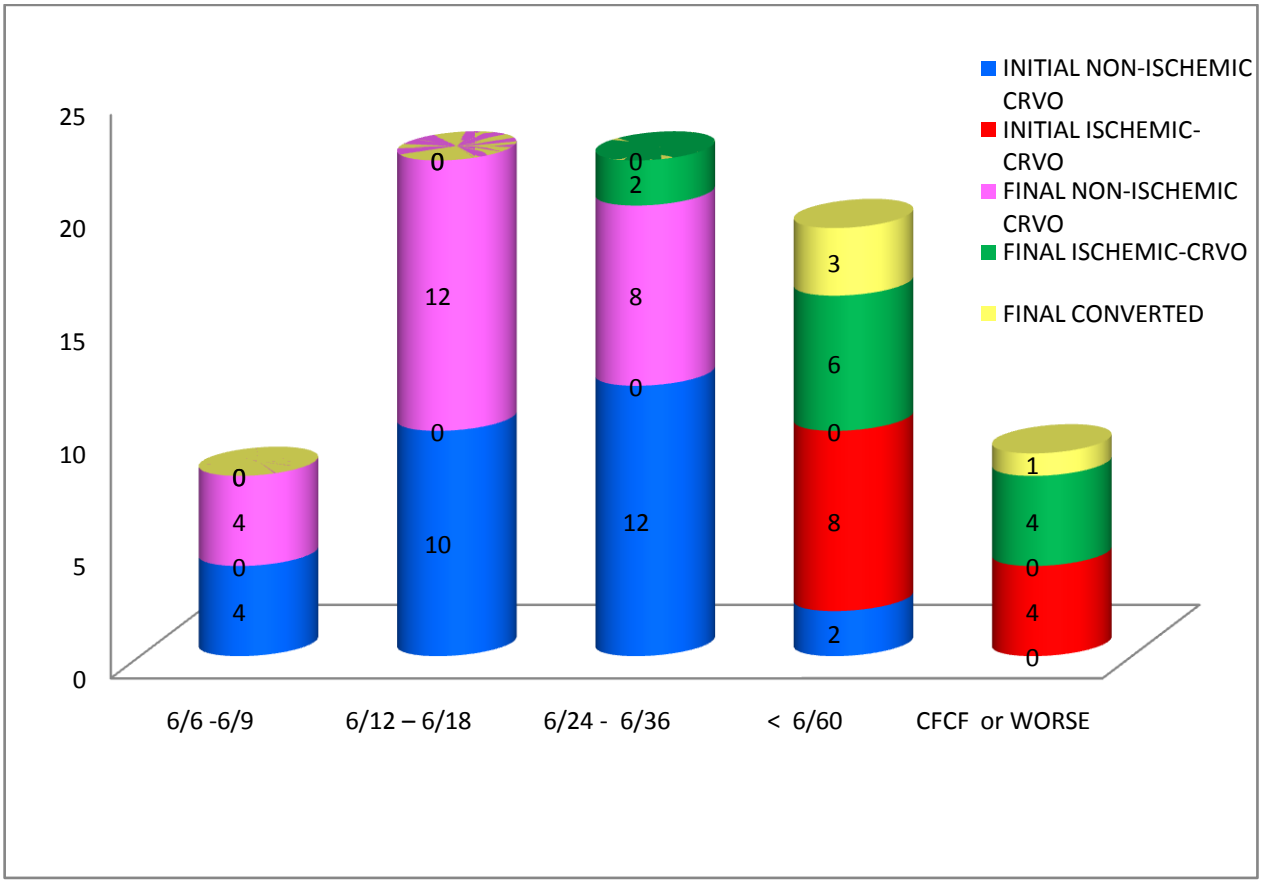
CLINICAL MANIFESTATIONS

CHART 8



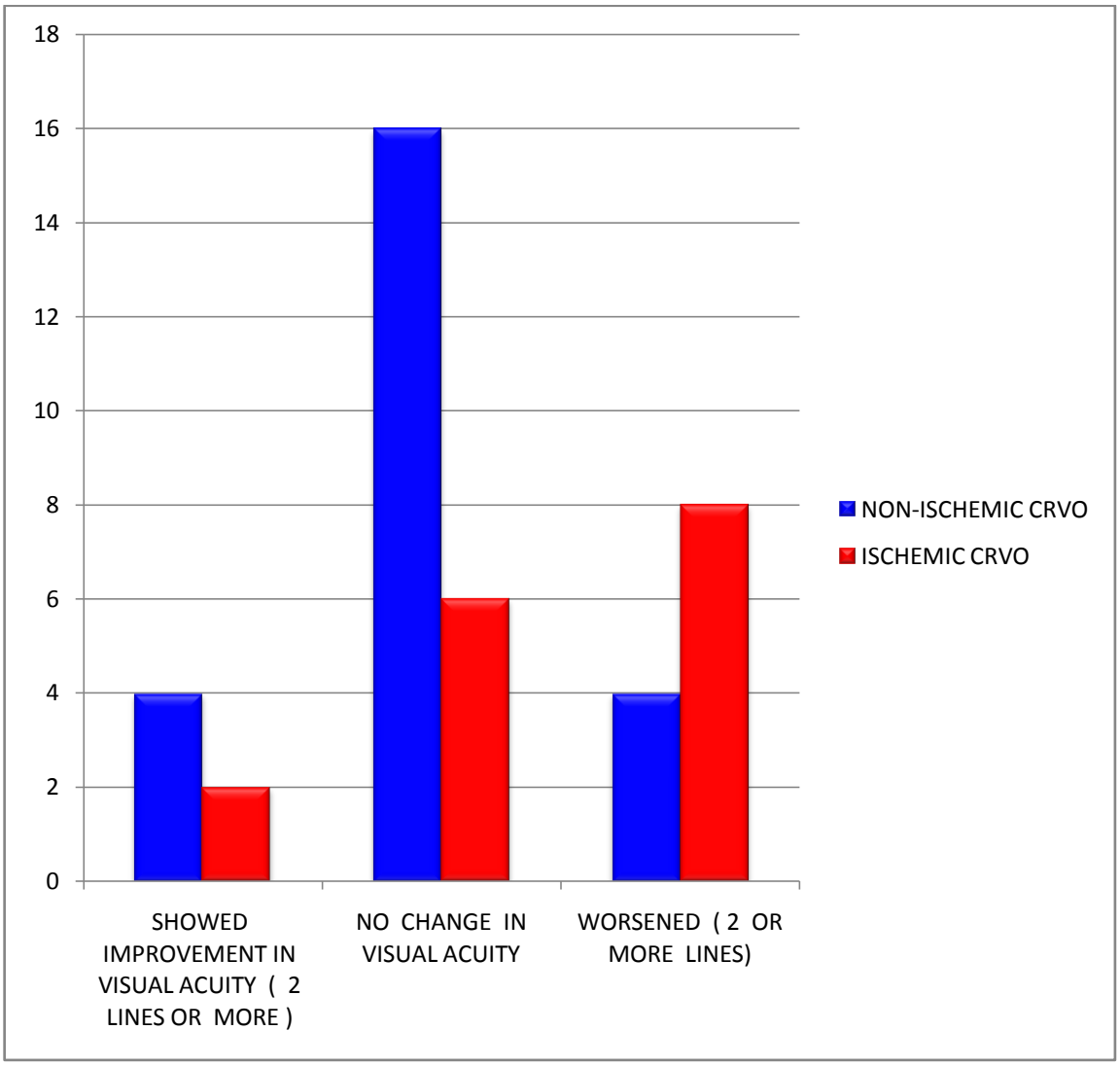
VISUAL ACUITY

CHART 9



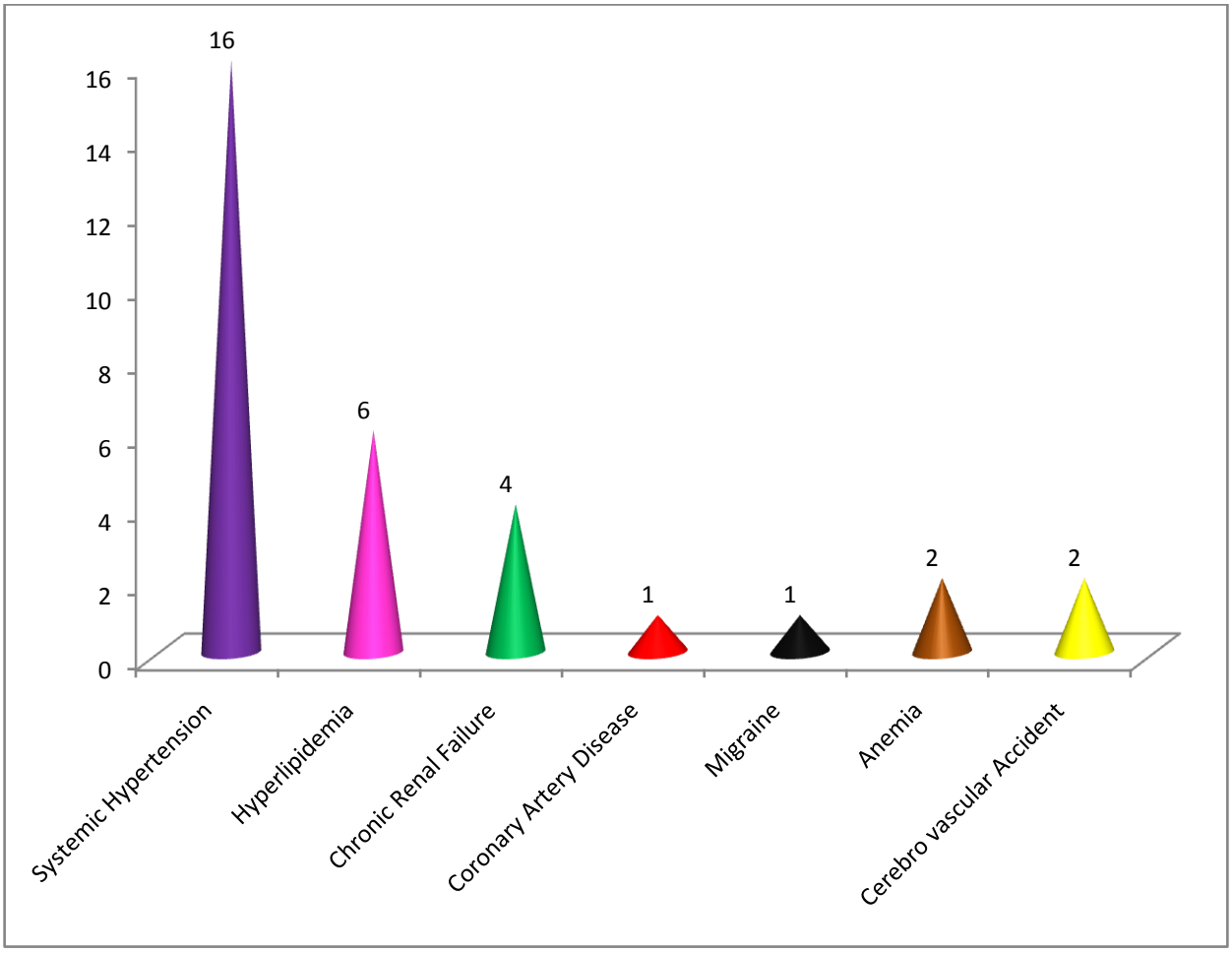
VISUAL ACUITY

CHART 10



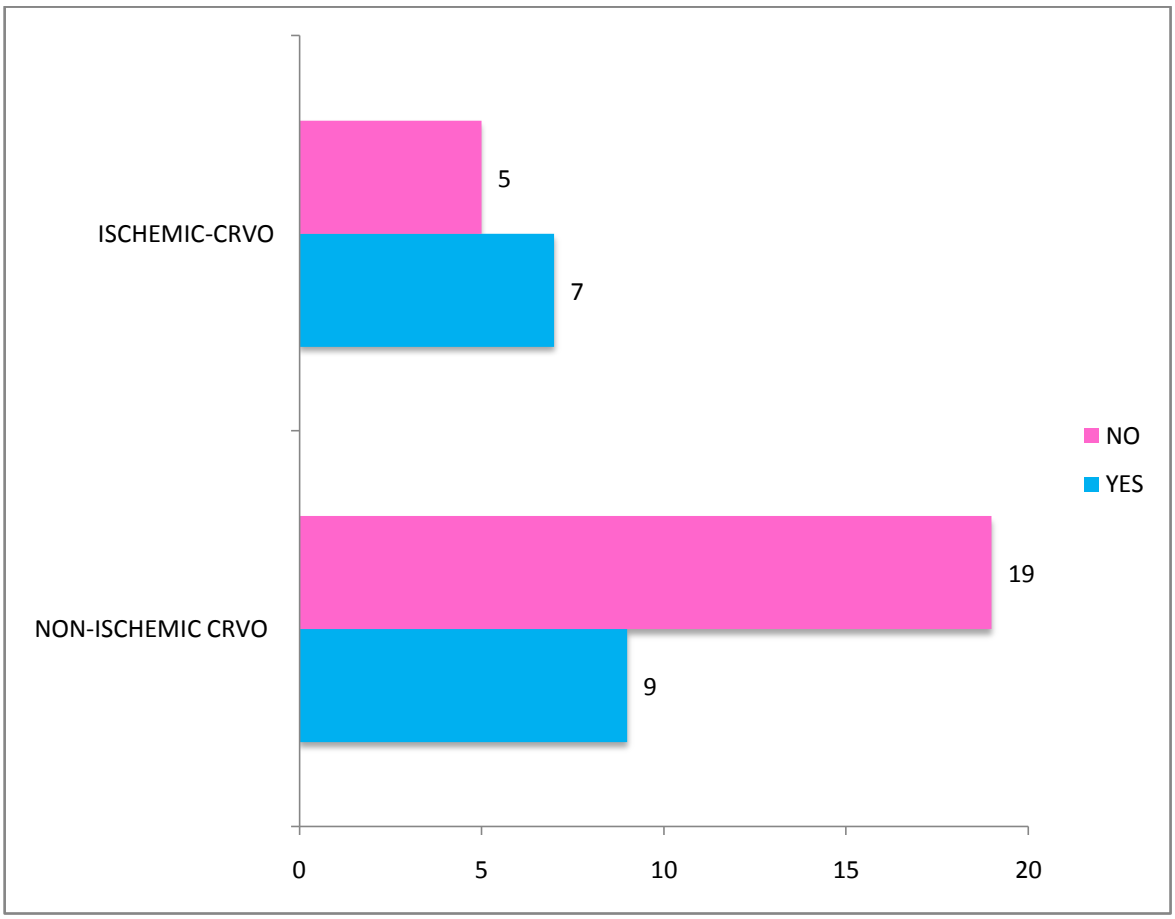
ASSOCIATED MEDICAL CONDITIONS

CHART 11



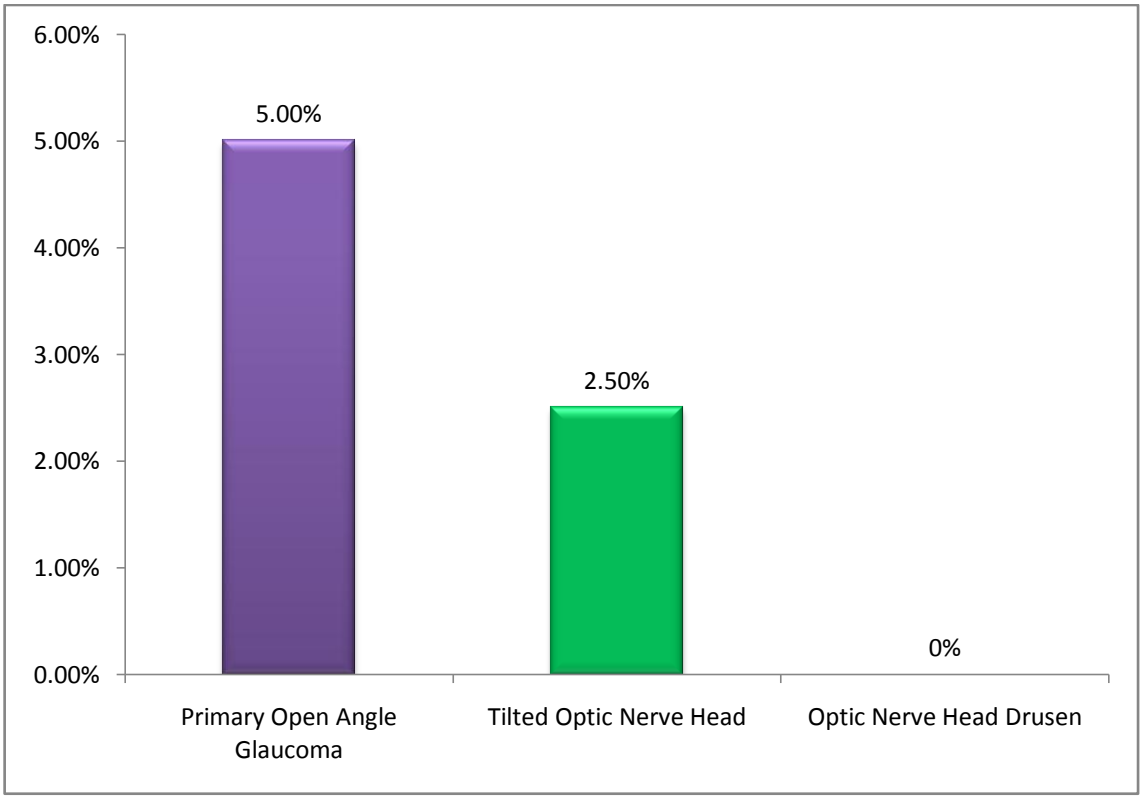
COMPARISION OF FREQUENCY OF HYPERTENSION IN NON ISCHEMIC AND ISCHEMIC - CRVO

CHART 12



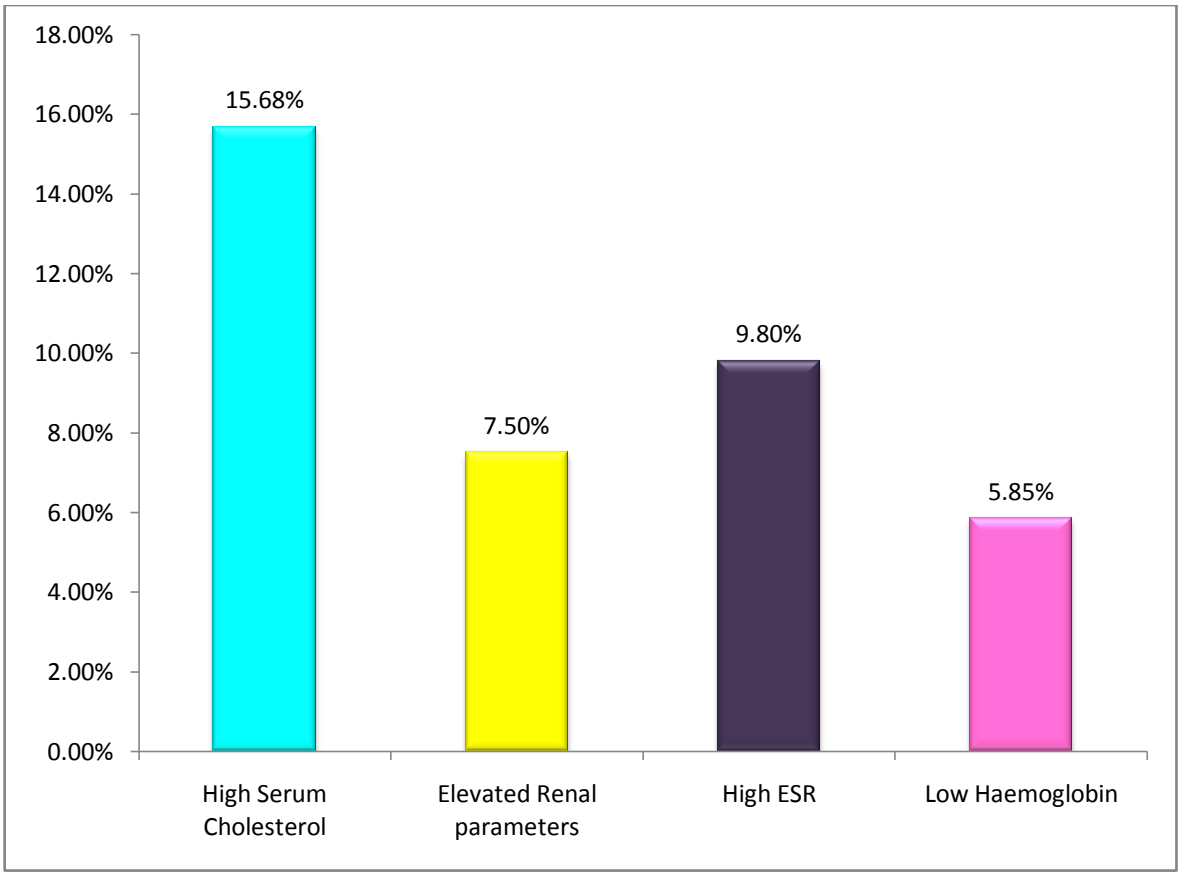
ASSOCIATED OCULAR CONDITIONS

CHART - 13



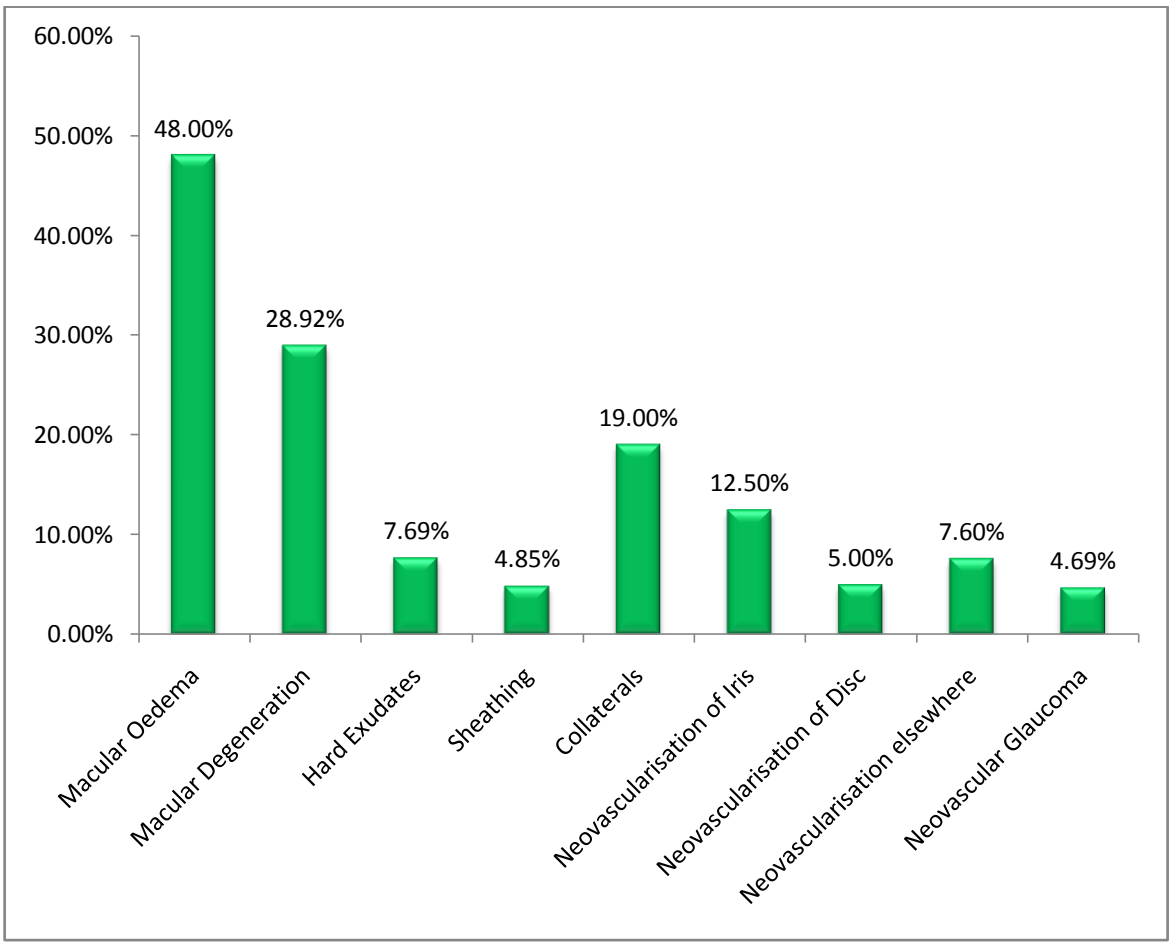
RESULTS OF LAB STUDIES

CHART 14



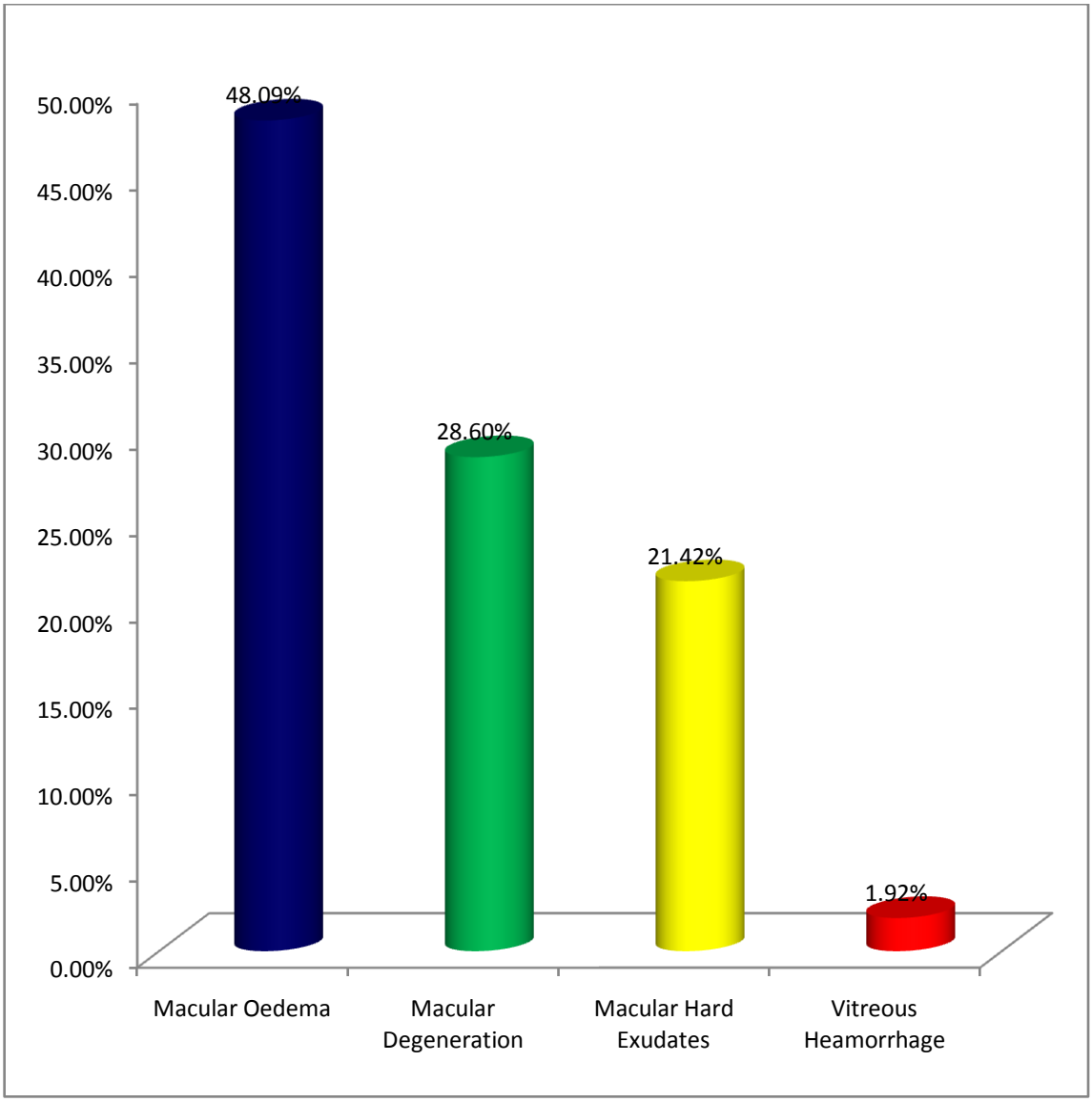
OCULAR SEQUELAE

CHART 15



CAUSES OF POOR VISUAL RECOVERY

CHART 16

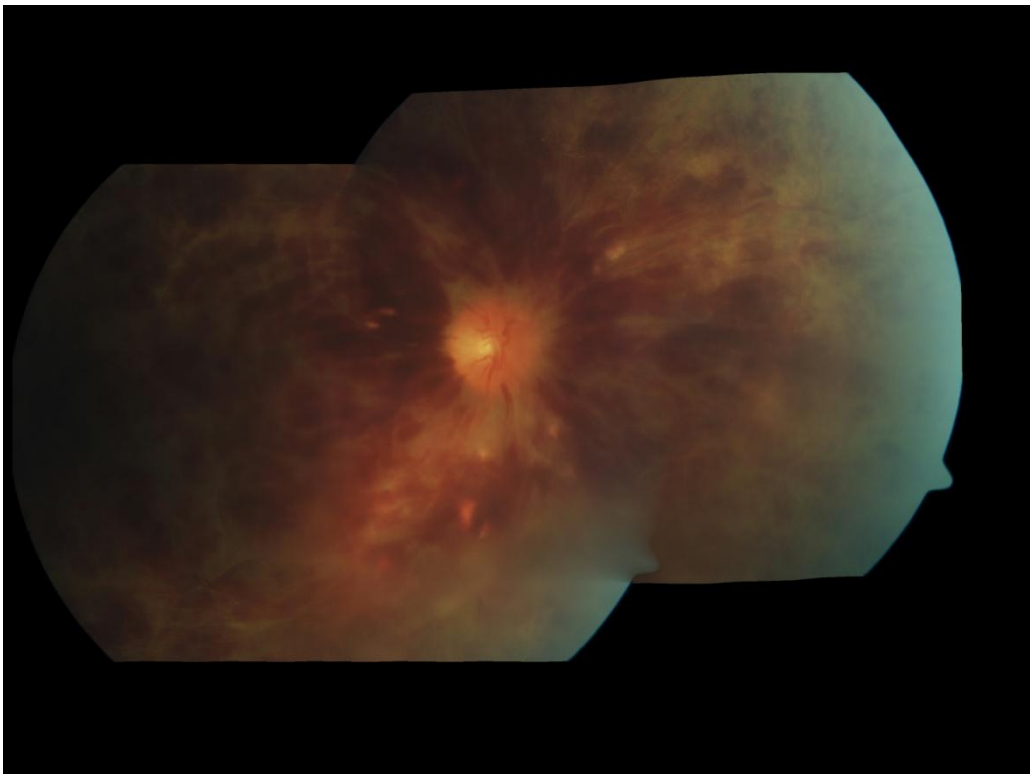


ANNEXURE

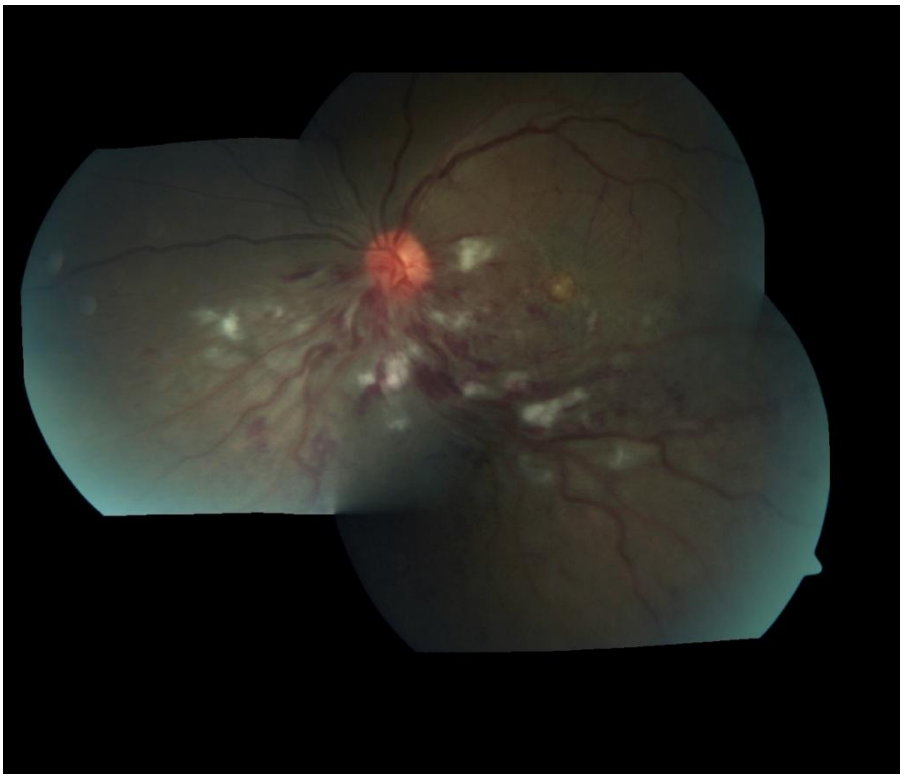
CRVO	-	Central Retinal Vein Occlusion
HCRVO	-	Hemi Central Retinal Vein Occlusion
BRVO	-	Branch Retinal Vein Occlusion
HCY	-	Homocysteine
SLE	-	Systemic Lupus Erythematosus
OR	-	Odds Ratio
C.I	-	Confidence Intervals
RVO	-	Retinal Vein Occlusion
CFCF	-	Counting Fingers Close to Face

NON – ISCHEMIC CRVO

ISCHEMIC CRVO



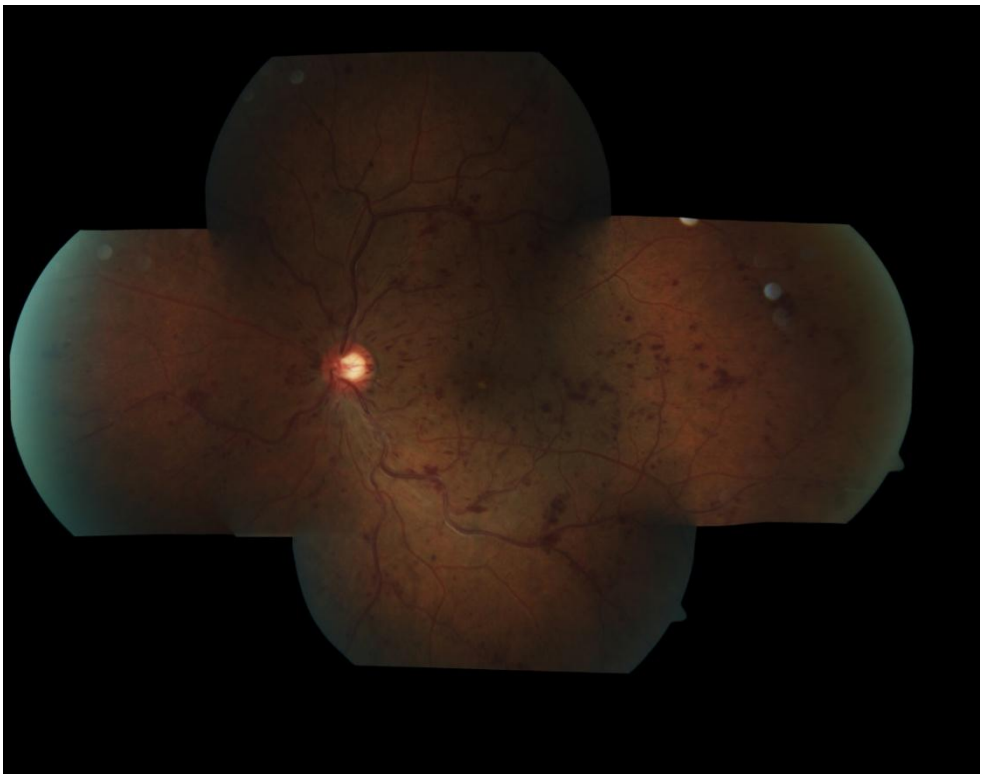
BRVO

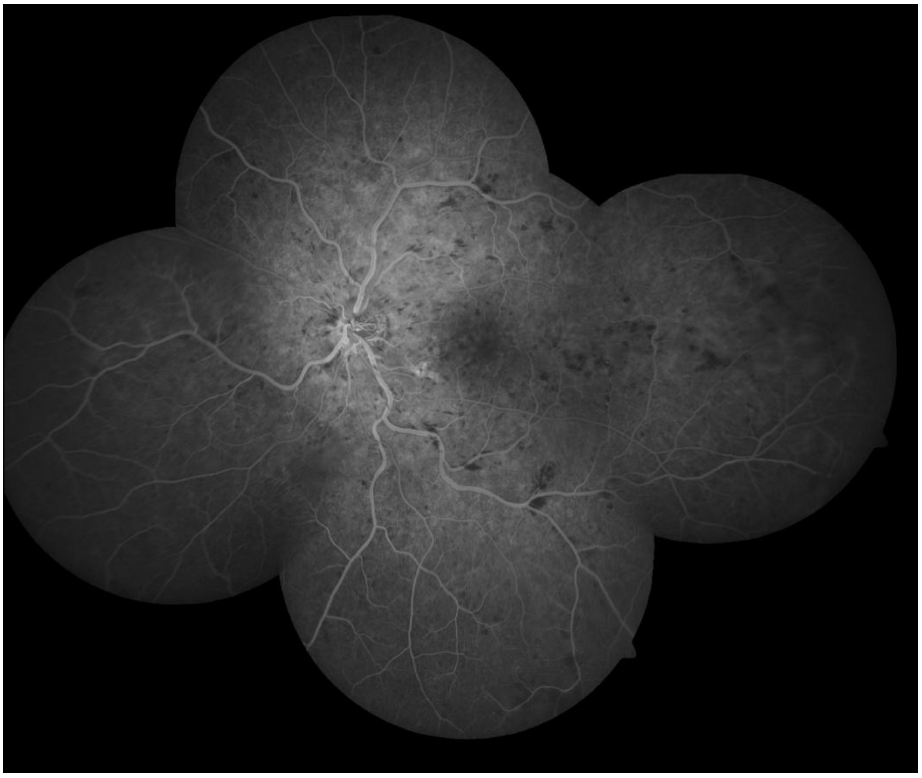


HCRVO

NON – ISCHEMIC CRVO

Paraman – 58/ Male

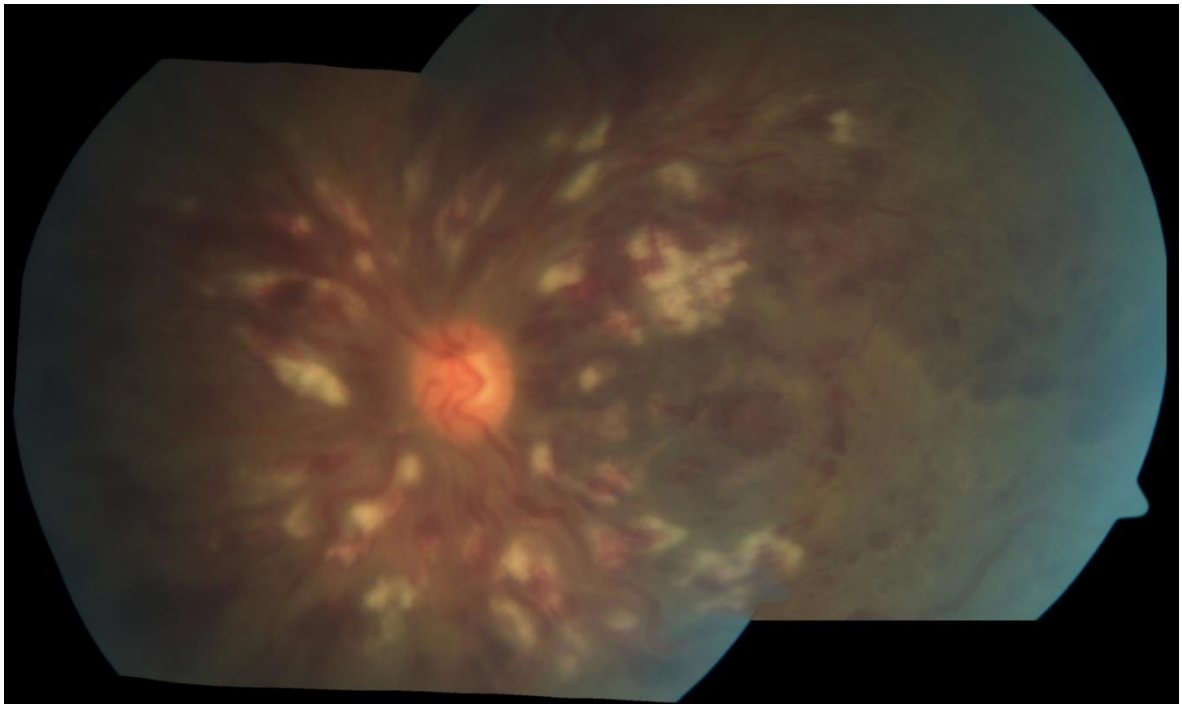


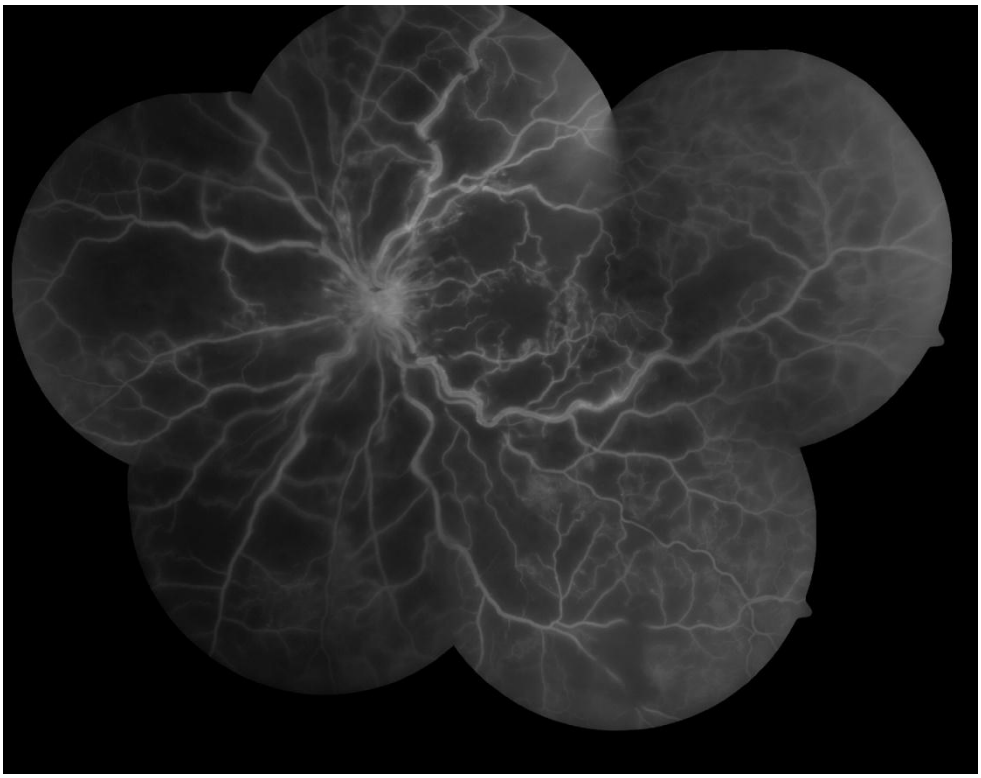


FFA PICTURE

ISCHEMIC CRVO

Ayyavu 68/ Male





FFA picture

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PROFORMA

Name : Age Sex

OP No.

History:

- ❖ Chief Complaints
  - Duration
    - ↗ Less than 6 months
    - ↘ More than 6 months
- ❖ History of Presenting illness
- ❖ Past History: Yes No

  Duration
  - H/o Hypertension
  - H/o Coronary artery Disease
  - H/o Chronic Renal failure

    (Renal Disease)
  - H/o Malignancy

- H/o Vascular Occlusive events Yes No

Duration

Eg : Cerebro Vascular Infarcts

- H/o Chronic Liver Disease
- H/o Drug Intake
- H/o Trauma
- H/o Previous Intra ocular Surgery
- H/o Previous Photo coagulation Procedures
- H/o Migraine
- H/o Oral Contraceptive usage (in femals)
- Chronic illness like tuberculosis, HIV

❖ Personal H/o Yes No

Duration

- Smoking
- Alcohol
- Physical Activity

❖ Diagnosis:

❖ Eye:

1. Right
2. Left
3. Both

❖ Ocular Examination (Both Eyes)

1. Visual Acuity
   - Best Corrected — Distance / Near
2. Visual fields
3. Pupillary reaction 1.Normal 2.Abnormal
4. Introcular pressure
5. Anterior Segment – Examination
   - Slit lamp Examination
   - Gonioscopy
6. Posterior Segment
   - Slit lamp Biomicroscopy with +90 D
   - Indirect ophthalmoscopy.
7. Fundus flourescein angiography
8. Fundus Photographs

Follow up

Ref. No. 3104/E4/3/2012 Govt.Rajaji Hospital,Madurai.20.
Dated: .03.2012

Institutional Review Board / Independent Ethics Committee.

Dr. A. Edwin Joe, M.D (FM), BL.,
Dean, Madurai Medical College & 2521021 (Secy)
Govt Rajaji Hospital, Madurai 625020.
Convenor
grhethicssecy @gmail.com.

Sub: Establishment-Govt. Rajaji Hospital, aMadurai-20-
Ethics committee-Meeting Agenda-communicated-regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held at 11.00 Am to 1.00Pm on 29.03.2012 at the Dean Chamber, Govt. Rajaji Hospital, Madurai. The following members of the committee have been attended the meeting.

1. Dr.N.Vijayasankaran,M.ch(Uro.) 094-430-58793 0452-2584397	Sr.Consultant Urologist Madurai Kidney Centre, Sivagangai Road,Madurai	Chairman
2. Dr.P.K. Muthu Kumarasamy, M.D., 9843050911	Professor & H.O.D of Medical, Oncology(Retired)	Member Secretary
3. Dr.T.Meena,MD 094-437-74875	Professor of Physiology, Madurai Medical College	Member
4. Dr. S. Thamilarasi, M.D (Pharmacol)	Professor of pharmacology	
5. Dr.Moses K.Daniel MD(Gen.Medicine) 098-421-56066	Professor of Medicine Madurai Medical College	Member
6. Dr.M.Gobinath,MS(Gen.Surgery)	Professor of Surgery Madurai Medical College	Member
7. Dr.S. Dilshadh, MD(O&G) 9894053516	Professor of OP&Gyn Madurai Medical College	Member
8. Dr.S.Vadivel Murugan., M.D, 097-871-50040	Professor of Medicine Madurai Medical College	Member
9. Shri.M.Sridher,B.sc.B.L. 099-949-07400	Advocate, 2, Deputy collectors colony 4th street KK Nagar, Madurai-20.	Member
10. Shri.O.B.D.Bharat,B.sc., 094-437-14162	Businessman Plot No.588, K.K.Nagar,Madurai.20.	Member
11.Shri. S.sivakumar,M.A(Social) Mphil 093-444-84990	Sociologist, Plot No.51 F.F, K.K Nagar, Madurai.	Member

Following Projects were approved by the committee

Dept of Opthal

Sl. No	Name of P.G.	Course	Name of the Project	Remarks
1.	Hema. D	PG, M.S (Ophthal)	Clinical study of central retinal vein occlusion in non-diabetics	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. She/He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. She/He should not deviate for the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events pr Serious adverse reactions.
4. She/he should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and apply for if any Extension of time is required She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the word or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

DEAN

To
All the above members and Head of the Departments concerned.
All the Applicants.

MASTER CHART

S.NO	NAME	AGE	SEX	OP. NO	TYPE OF CRVO	LATERALITY	VISUAL ACUITY		SYSTEMIC DISEASES	CLINICAL MANIFESTATIONS
							INITIAL	FINAL		
1	KOORU AMBALAM	47	M	536956	NON-ISCHEMIC	RIGHT EYE	6/18	6/12	HYPERLIPIDEMIA	SUDDEN PAINLESS LOSS OF VISION,BLURRING OF VISION
2	SONAIMUTHU	52	M	546800	NON- ISCHEMIC	LEFT EYE	6/36	5/60	-	SUDDEN PAINLESS LOSS OF VISION, FLOATERS
3	MUTHU KALAI THEVAR	62	M	521364	ISCHEMIC	LEFT EYE	5/60	6/24	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION, BLURRING OF VISION, FLOATERS
4	KARUPAYEE	49	F	510482	NON-ISCHEMIC	LEFT EYE	6/24	6/12	-	BLURRING OF VISION, FLOATERS
5	RAJAKARUTHAI	56	M	536804	NON-ISCHEMIC	RIGHT EYE	6/36	6/18	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION, FLASHES OF LIGHT
6	SEKAPI	46	F	516809	NON-ISCHEMIC	LEFT EYE	6/6	6/9	HYPER LIPIDEMIA	BLURRING OF VISION, SUDDEN PAINLESS LOSS OF VISION
7	BEER MOHAMMED	68	M	540028	NON-ISCHEMIC	LEFT EYE	5/60	2/60	CORONARY ARTERY DISEASE	SUDDEN PAINLESS LOSS OF VISION, AMAUROSIS FUGAX.
8	JEGATHEESWARAN	42	M	504220	NON-ISCHEMIC	RIGHT EYE	6/12	6/9	HYPER LIPIDEMIA	BLURRING OF VISION, SUDDEN PAINLESS LOSS OF VISION
9	RAMAIH	50	M	561284	NON-ISCHEMIC	RIGHT EYE	6/18	6/12	SYSTEMIC HYPERTENSION	BLURRING OF VISION
10	OTCHA THEVAR	65	M	533602	ISCHEMIC	LEFT EYE	2/60	6/60	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION, RAPD PRESENT

11	VEERA LAKSHMI	65	F	534024	ISCHEMIC	RIGHT EYE	3/60	6/60	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION, RAPD PRESENT
12	RAMAR	52	M	520016	NON-ISCHEMIC	RIGHT EYE	6/18	6/24	-	BLURRING OF VISION
13	PARAMAN	58	M	510985	NON-ISCHEMIC	RIGHT EYE	6/36	6/18	SYSTEMIC HYPERTENSION	BLURRING OF VISION, SUDDEN PAINLESS LOSS OF VISION
14	PITCHAIKAL	40	M	551842	NON-ISCHEMIC	LEFT EYE	6/12	6/9	CHRONIC RENAL FAILURE	SUDDEN PAINLESS LOSS OF VISION
15	KATHAYEE	48	F	560426	NON-ISCHEMIC	LEFT EYE	6/9	6/18	HYPER LIPIDEMIA	SUDDEN PAINLESS LOSS OF VISION
16	MUTHUSAMY	48	M	551265	NON-ISCHEMIC	RIGHT EYE	6/24	6/36	-	BLURRING OF VISION
17	PANCHAVARNAM	60	F	509874	NON-ISCHEMIC	RIGHT EYE	6/24	6/36	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION; FLOATERS PRESENT
18	AYYAVU	68	M	546894	ISCHEMIC	LEFT EYE	1/60	4/60	CERBRO VASCULAR ACCIDENT	SUDDEN PAINLESS LOSS OF VISION; RAPD PRESENT
19	SELVARAJ	49	M	538250	NON-ISCHEMIC	LEFT EYE	6/18	6/12	-	BLURRING OF VISION.
20	RAJAMMAL	56	F	504862	NON-ISCHEMIC	LEFT EYE	6/36	6/36	SYSTEMIC HYPERTENSION	FLOATERS PRESENT.
21	ABUKKAR	56	M	566891	NON-ISCHEMIC	RIGHT EYE	6/9	6/12	SYSTEMIC HYPERTENSION	BLURRING OF VISION
22	MUTHURAMAN	52	M	516250	ISCHEMIC	RIGHT EYE	5/60	6/36	CHRONIC RENAL FAILURE	SUDDEN PAINLESS LOSS OF VISION,FLOATERS PRESENT

23	LAKSHMANAN	62	M	584050	NON-ISCHEMIC	RIGHT EYE	6/60	HM+	CERBRO VASCULAR ACCIDENT	AMAUROSIS FUGAX; SUDDEN PAINLESS LOSS OF VISION
24	PERUMAYEE	61	F	526324	ISCHEMIC	LEFT EYE	4/60	1/60	MIGRAINE	SUDDEN PAINLESS LOSS OF VISION, RAPD PRESENT
25	PANDIAN	70	M	586020	ISCHEMIC	RIGHT EYE	PL,PR +	PL,PR+	CHRONIC RENAL FAILURE	SUDDEN PAINLESS LOSS OF VISION, RAPD PRESENT
26	SOMU	58	M	594274	NON-ISCHEMIC	RIGHT EYE	6/24	6/24	-	FLOATERS PRESENT
27	CHINNA SAMY	72	M	506270	ISCHEMIC	LEFT EYE	CFCF	PL,PR+	SYSTEMIC HYPERTENSION	BLURRING OF VISION,RAPD PRESENT
28	KARUNAKARAN	59	M	514254	NON-ISCHEMIC	LEFT EYE	6/12	6/24	HYPER LIPIDEMIA	BLURRING OF VISION, FLOATERS PRESENT
29	VELAMMAL	69	F	556274	NON-ISCHEMIC	LEFT EYE	6/36	5/60	ANEMIA WITH SYSTEMIC HYPERTENSION,	SUDDEN PAINLESS LOSS OF VISION, FLOATERS
30	THANGAVEL	62	M	573240	NON-ISCHEMIC	RIGHT EYE	6/18	6/12	ANEMIA	SUDDEN PAINLESS LOSS OF VISON, FLOATERS
31	RAMAIYA THEVAR	69	M	566220	ISCHEMIC	LEFT EYE	3/60	6/60	SYSTEMIC HYPERTENSION	BLURRING OF VISION WITH RAPD
32	MANIKANDAN	68	M	570238	ISCHEMIC	LEFT EYE	1/60	5/60		SUDDEN PAINLESS LOSS OF VISION.
33	KALIAPPAN	62	M	524082	NON-ISCHEMIC	RIGHT EYE	6/12	6/12	SYSTEMIC HYPERTENSIO	BLURRING OF VISION
34	MUNIYANDI	67	M	535082	ISCHEMIC	LEFT EYE	HM+	PL,PR+	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION,RAPD PRESENT
35	KATHAYEE	62	F	540875	NON-ISCHEMIC	RIGHT EYE	6/36	6/12	HYPER LIPIDEMIA	AMAUROSIS FUGAX

36	ALAGAR SAMY	60	M	560891	ISCHEMIC	LEFT EYE	CFCF	HM+	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION
37	CHANDRAN	56	M	52616	NON-ISCHEMIC	LEFT EYE	6/24	6/60	-	BLURRING OF VISION, FLOATERS
38	VELLAI SAMY	60	M	531842	NON-ISCHEMIC	RIGHT EYE	6/9	6/24	CHRONIC RENAL FAILURE	SUDDEN PAINLESS LOSS OF VISION
39	MARUTHAN	69	M	540265	NON-ISCHEMIC	LEFT EYE	6/36	5/60	SYSTEMIC HYPERTENSION	SUDDEN PAINLESS LOSS OF VISION, FLOATERS
40	RAKKAYEE	58	F	597248	NON-ISCHEMIC	LEFT EYE	6/18	6/18	-	BLURRING OF VISION

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